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VOLUME 1 NUMBER 2

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

TUBERCULOUS MENINGITIS

AT the last clinic we discussed the general subject of tuberculosis in infancy. Let us devote this hour to a consideration of tuberculous meningitis.

Tuberculous meningitis in children presents itself in many forms and under many guises. If students would observe a large series of cases (100, 200, or 300) they would be impressed with the wide variation of onsets, symptoms, course, duration, termination, and even the findings.

Here is a little girl eleven and a half years old; note her position on the table: her knees flexed; thighs somewhat drawn on the abdomen; legs flexed somewhat on the thighs. One commonly sees this posture in a well-developed tuberculous meningitis. When lying flat on the back there is more or less opisthotonus and rigidity of the muscles of the neck.

(Patient brought in. History read.)

History.—Doris H., aged 11½ years, was admitted to Michael Reese Hospital March 11th, with a history of headache, vomiting, extreme constipation, weakness, anorexia, and extreme thirst. Onset a week ago, when patient appeared listless and sleepy. In school she rested her head on the desk and tried to sleep, so was sent home. She was extremely constipated, which she had never been before. She complained of a headache, frontal in type and throbbing in character. She has vomited nearly every day since the onset, occurring usually imme-

diately after eating or drinking, projectile in type. She has never been delirious, but her mother says she has been very irritable for the past week.

Previous illnesses: Measles at $2\frac{1}{2}$ years, scarlet at 6 years, diphtheria at 8 years, pneumonia at 3 years, also chicken-pox and whooping-cough.

Family history is negative.

Laboratory findings:

1. Urine—Negative.
2. Blood—Hb. 80 per cent.

Wbc.	5400	11th
	8400	12th
	8200	13th
	8600	14th

Rbc. 4,265,000

Differential: N., 58 per cent.; SM., 20 per cent.; LM., 12 per cent.

3. Widal—Negative 12th and 13th.
4. Blood-culture—Negative, forty-eight hours.
5. Spinal puncture—Clear fluid. Direct smear, no organism.

   Direct count—320 per c.mm.

   Noguchi—Strongly positive.

   Ross Jones—Strongly positive.

   Nonne—Negative.

   Differential—98 per cent. lymphocytes; 2 per cent. polymorphs.
6. Von Pirquet—Positive.

DOCTOR: What is your name?

PATIENT: Doris.

DOCTOR: Do you go to school, Doris?

PATIENT: Yes.

DOCTOR: Who is your teacher?

PATIENT: I have forgotten the name of the teacher.

DOCTOR: Do you like her very much? Do you like your teacher?

PATIENT: Not so well.

DOCTOR: Does your head ache, Doris?

PATIENT: Yes.

DOCTOR: Where?

(Patient indicates forehead.)

DOCTOR: What is your best study in school?

PATIENT: Spelling.

DOCTOR: She is a bright little girl. Open your eyes wide, Doris. You will notice that there is a slight difference in the palpebral fissures, which is apparent at this moment. She is, for the most part, pale, but there is a slight flush over the cheek-bones. Note that she lapses into sleep while we examine her. At this moment both pupils are dilated and equal. On a previous examination a striking inequality of the pupils was observed, the left being larger than the right.

The neck is quite rigid, and if one attempts to lift the child by raising the head one notices that almost the entire back, especially the cervical and dorsal regions, are raised from the table. (Demonstrates the rigidity.) The lips are dry, with herpes on both upper and lower lip.

The pulse is slow, not the inhibition pulse that we sometimes observe. The pulse, as the record shows, is irregular in time. Yesterday's record shows that it was as low as 88 and as high as 108. The temperature yesterday was as low as 102° F. and as high as 104° F. She began at 4 o'clock with a temperature of 102.2° F., and at 8 o'clock her temperature was 104° F.

The tongue is coated. It shows a distinct fine tremor and it deviates toward the right side.

You notice that she is emaciated; she looks thin; she shrinks; she objects to being touched; she evidently has some hyperesthesia. By stroking her body one notices that the skin very rapidly is affected; a red flush appears where the finger-nail has been drawn across it. Can you see it?

VISITOR: Yes, it is very marked. The abdomen is sunken in; concave. (Patient weeping.) The liver is not palpable.

DOCTOR: Note the position in which she lies, the thighs drawn up toward the abdomen. Now if we flex the thigh decidedly and then attempt to make flexure of the leg on the

thigh we find that flexion is impossible—a positive Kernig's sign. The left leg shows the same phenomenon. I should say that it was about equal on both sides. So that we have in this case a positive bilateral Kernig's sign.

If we place one hand under the child's head and the other hand over the thorax and then raise the hand beneath and watch the position and condition of the feet and the legs, what happens? She draws up her legs suddenly at the knees; the toes separate, fan-like. Both legs are similarly affected. Watch this again. (Demonstration repeated.)

She bends her legs slightly at the knees. The feet tend to evert, and there is slight, not very marked, separation of the toes. This is Brudzinski's neck sign.

Upon tapping the face over the malar bone no muscular twitching occurs. In consequence, we may say that the facial sign, known as Chvostek's sign, is absent.

The knee reflex is present on both sides, though not very active.

(Doctor strokes the sole of child's foot with a match.) What is this? The large toe goes upward and the other toes tend to spread slightly and go downward.

Now try the right side. You notice the same phenomenon. Now watch—a positive Babinski. So, then, we have a confirmation of our suspicions at the outset that the child is suffering from meningitis. We may say, then, that a positive Babinski phenomenon consists of the simultaneous extension of the big toe with a spreading and flexion of the other toes when the sole of the foot is stroked.

On the left arm here we have a moderately positive von Pirquet reaction; not striking, nevertheless present. All these signs and the peculiar psychic condition of the child—the hyperesthesia, the attitude of resentment at being turned, disliking to be touched, the irregularity of the pulse, the inequality of the pupils—lead us to infer that the child is suffering from tuberculous meningitis. It will develop in the course of the discussion that our diagnosis is confirmed by the prodroma and peculiarity of onset, the study of the spinal fluid, and the clinical course.

I want to consider in some detail the mode of etiology and the pathogenesis. Some one suggested that tuberculous meningitis is more common in some years than in others. We know this is true of certain diseases. We know, for example, that last year as well as this year this city and possibly the entire North or Middle Western country was overwhelmed with measles. We know as a matter of experience that some years there are fewer cases than during the epidemic years. So it is of other acute contagious diseases, especially scarlet fever. Some years it occurs in veritable epidemics; in other years it is merely sporadic. It is true of other diseases. Cerebrospinal meningitis seems to come in a certain cycle. Some years great numbers of individuals are attacked. Great epidemics have been reported that stretched from one end of the country to the other. The same is true of anterior poliomyelitis. But it is a question if this applies to tuberculous meningitis. Statistical investigation seems to show that tuberculous meningitis does not vary so much from year to year, but its occurrence seems to bear a striking relationship to measles. When measles is raging, then we may expect a large mortality from tuberculous meningitis to follow. The same statement may be applied to whooping-cough. The occurrence of tuberculous meningitis has a definite seasonal variation. We know very well that when the spring of the year comes, at about this time, March and April, and even May, the greatest number of cases of tuberculous meningitis are admitted to the hospital. Every clinician knows that. But why? Why there should be more cases of tuberculous meningitis in the early spring is difficult to say, except that perhaps during the winter the acute respiratory diseases seem to influence somewhat the activity of the tuberculous process in the body, tending to render active a latent tuberculosis. Then possibly, too, the unfavorable conditions of hygiene and general living during the winter, the fact that children are kept closed up in-doors, that the living conditions conducive of health are less favorable during the long cold winter than during the summer, must be considered as important factors. Perhaps these in some way influence the seasonal occurrence, but it is true that from

January to July we see the largest number of cases of tuberculous meningitis; the largest number occur during the month of April; the smallest number occur between the months of August and December. I am quoting from Herbert Koch's statistics which were gathered from the Vienna Clinics. His statistics show that 61 per cent. occurred from January to July; that 39 per cent. occurred during the last six months of the year, and the very fewest cases, only 5 per cent., during November and December. These statistics are important for students of this disease to know.

Age has an enormous influence on the occurrence of the disease. The largest number of cases of tuberculous meningitis occur during the first four years of life, and the greatest number occur during the second year. One would think that the largest number of cases would occur during the first year. This would apparently follow from what we said during the previous hours, namely, that tuberculosis, like other infectious diseases, makes severe ravages on account of the low resistance of the babies, and the mortality is high; but then, on the other hand, we know that a great deal of tuberculosis mortality during the first year of life is from other types than meningitis.

Now, do not misunderstand me, that tuberculous meningitis does not occur during the first year, because if it were necessary we could go to our records and exhibit history sheets of babies that died during the first year from tuberculous meningitis. However, statistics show that by far a larger number of infants die during the second year from tuberculous meningitis.

What influence has heredity on tuberculous meningitis? Heredity may play a part; heredity contributes slightly toward the propagation of tuberculosis from parent to child; we know that the most important factor in tuberculous meningitis is the presence of tuberculosis in some adult member or members of the family, and we believe that in maternal tuberculosis the contagion is carried from mother to baby after birth, possibly long after birth. Occasionally, too, there are several children in the same family who die from tuberculous meningitis. We know from our records that parents come in here and tell us with

great apprehension that they have already lost one child from this disease or from a similar condition, and they are fearful that this child is also afflicted. I know in my own practice of families where several children have died from meningitis, and Koch tells us that in his abundance of material he found a family in which 4 children died from tuberculous meningitis.

To summarize, then, we may say that hereditary transmission in tuberculous meningitis is not the general rule; we may look upon it as an incident, due possibly to the fact that tuberculosis is easily conveyed in a certain environment.

Another question to be considered is whether boys and girls are affected with the same frequency, and all reliable statistics show that there is no difference. Boys and girls are equally affected; there is no preponderance of one sex over the other.

We have spoken about maternal nursing, and you remember that we do not advise a tuberculous mother to nurse her baby. We always say that open tuberculosis is a positive and absolute contraindication to nursing. We accept that as conclusive. All authorities agree to it, and if we need any support for this view we might go to the statistics, which prove that babies nursed at the breast are not spared from tuberculous meningitis. Indeed, the startling revelation is that 86 per cent. of all cases of meningitis occur in breast-fed children. These are the Vienna statistics. The figures are in a sense somewhat contradictory. We teach and we believe that breast feeding is most healthful, increases the resistance, and lessens morbidity and mortality. An analysis of the statistics shows that tuberculous meningitis occurs with great frequency in breast-fed children, which implies close, intimate contact with the mother.

I have already said that measles bears a striking relation to the occurrence of tuberculous meningitis. How many, many times you will hear a mother say that the baby had measles at eight months or ten months and tuberculous meningitis at a year or fourteen months or even earlier. Now just what that means does not appear at a glance. Why should measles predispose to tuberculous meningitis? Is it possibly because of the acute respiratory involvement associated with measles or the enanthem,

or the eruption which is produced in the larynx or on the surface of the mucous membrane of the bronchi? Possibly the inflammation on the mucous surfaces stimulates a latent process to become an active one. Or is it a more subtle cause, which the philosophic, speculative minds of the laboratory workers must solve more fully? It is hinted that in some way or other the resistance of the body is much lowered on account of the reduction of the antibodies, when general infection by the tubercle bacilli is invited or becomes possible.

Trauma sometimes has a relation to the occurrence of tuberculous meningitis. It is well to discount trauma when you take the history of the baby, but still you should always give the patient the benefit of every reasonable doubt. Let me say in passing, don't make light of the story of the mother if she is talking pointedly, if she is telling facts. Very often the mother wanders, she is excited, she may tell things that have no relation to the matter in hand; if she is talking straightforwardly, listen patiently. As far as trauma is concerned, nearly every baby gives a history of injury: nearly every baby has fallen out of bed; nearly every baby has bumped its head somewhere; but, on the other hand, severe trauma seems to bear some relation to the occurrence of this disease.

I remember very well an older baby, two years or two years and a half old, presented some evidence of an involvement of the hip-joint. The condition had not progressed very far; there was something suggestive in the gait, some indefinite suggestion of pain; later on the baby fell out of a window and fractured a thigh; in a little while there was a secondary reaction and we saw very distinctly that there was fluctuation, swelling, undoubtedly an acute tuberculous abscess in the thigh. The patient had not only fractured his thigh, but rendered a latent tuberculous process in the hip-joint an active one, and the localized process became a general disseminated tuberculosis. In the course of a short time his general demeanor changed, he became petulant, irritable, had considerable fever, and it was not a great while before the baby developed tuberculous meningitis and succumbed. These cases are not uncommon.

We have seen other cases that were operated on for tuberculous gland of the neck, with those large incisions by which the baby was cut wide open from the tip of the mastoid process to the end of nowhere, and where great dissections were made, great spaces laid wide open and tissues pretty thoroughly stirred up, so that access of the tubercle bacillus into the body was made easy. We know that tuberculous meningitis occurs after these operations.

As a rule, when tuberculous meningitis occurs it bears a close relation to some active tuberculous process in the body, frequently the lungs. At the autopsy of these babies you will find that the largest number of cases had an active tubercular process in the bronchial glands, or less commonly one finds old healed or calcified nodules in the bronchial glands. The point is, that most often tuberculous meningitis depends upon an active, florid tubercular focus somewhere in the body, most commonly in the bronchial gland.

Leaving the pathogenesis for a few moments, let me call your attention to some points in connection with the symptomatology:

In most of these cases of tuberculous meningitis the onset is gradual. In a large number of cases children have been manifesting for weeks or even months some change in demeanor; some change in their attitude toward life. In other cases the disease comes on suddenly, or apparently so. You know children react differently to disease. Some children will, in their subconscious nature, whimper and whine and complain, and exaggerate the slightest trouble, and other children, like adults, will stand up, show no signs, but act stoically, face death and danger without complaint. Some children are easy to handle, easy to examine, submit to it as if to the manner born, and others will resist as if engaged for a pugilistic contest. So it is difficult sometimes to interpret the findings and to determine the exact mental status and the attitude of the child. It is difficult for the physician and even harder for the parents to know how far the condition of irritability is one of disease or how far it is one of natural inborn cussedness.

Vomiting is characteristic, and according to the description of the old clinicians it is projectile; that is, it is easy, it occurs without any apparent effort, it occurs without any great nausea. There is no doubt that such vomiting is characteristic of acute tuberculous meningitis. Vomiting occurs easily; undoubtedly this means that there is some irritation or overstimulation of the center in the medulla.

Now the peculiar thing about the vomiting is that it will continue for several days in the beginning of the disease; sometimes it will last for a considerable time through the course of the disease, but most frequently the vomiting ceases when the disease becomes well established; in the larger number of cases it is an early symptom and indicates the period of cerebral irritation.

Children who are old enough to talk complain of headache. It is a peculiar thing about the localization of pain in infants and young children. They sometimes locate a pain very indefinitely. The baby may have a very severe headache and say his face hurts, his mouth hurts, his neck hurts, or that his chest hurts. In young children who are just beginning to talk the localization of pain is very indefinite and very inexact; older children complain of violent headache, and it is very often frontal, or they complain of a sense of fulness. Not only do they have spontaneous pain, but they complain if the head is touched. If it is tapped or percussed, they complain of a decided increase in the pain. This headache occurs not only early in the disease, but very often it continues through a considerable period of the illness. It may last a long time, even until unconsciousness occurs.

In addition to these symptoms, the patients very often show signs of restlessness. When in bed they throw themselves from side to side; they rarely lie on their backs; they moan; sometimes they localize the pain; at other times show a peculiar psychic condition which I will refer to in a moment. Very often they grind their teeth, making a disagreeable sound, and young infants frequently show a peculiar chewing movement, as though there was some irritation of the facial gustatory muscles.

Constipation so frequently occurs that it may be considered one of the most constant symptoms. You will remember that Doris, the little patient we have examined this morning, showed marked constipation. Children who have been previously normal as far as bowel movements are concerned, with the onset of the disease become constipated, obstinately constipated, so that even a strong cathartic will not move the bowels. Enemas act with great difficulty; undoubtedly, some atonic condition of the bowels causes a degree of intestinal paresis.

The psychic changes that occur in these children are of great interest. Little babies become, as I have explained to you, petulant, fretful, difficult to manage, restless night and day. Older children that have been normal, well behaved, are completely changed. They no longer want to come to father and mother, nor do they want to be caressed. They become resentful; they strike back; they say things that they never have been known to say before to those they love; they are completely changed. They reject the company of other children; they are not happy with their pets and toys; they do not care for anybody or anything; they simply want to be left alone. Sometimes a child in school completely changes in his attitude toward his studies. He loses ambition. He formerly learned readily; he was always ready to answer questions; he was always ready to recite and speak, and now he has lost interest.

This child, Doris, reclining her head on her desk, had no interest in what happened in the schoolroom, so the teacher sent her home. Many become depressed; then others become excited. I know one case particularly where the child was almost uncontrollable; stood up in bed; he acted like a wild animal; we could not approach him to examine him; we did not know what to make of him; we thought some acute insanity had come over him; he fought, bit, kicked, and made every kind of demonstration that a wild animal would make; finally he became calm, went into a comatose condition, and died of tuberculous meningitis. Occasionally at the very onset there is pronounced excitement.

Differential Diagnosis.—It is necessary at times to differentiate between tuberculous meningitis and other forms and con-

ditions. For example, encephalitis sometimes presents the greatest difficulties, appearing with all the meningocerebral symptoms that we are accustomed to find in true meningitis. High fever, more or less unconsciousness, and then the paralytic symptoms. In some cases it is not uncommon to find paralytic phenomena early in encephalitis; for example, hemiplegia is extremely common; that is, encephalitis of the superior type; while in encephalitis of the inferior type it is not uncommon to find a crossed paralysis indicating an involvement of the pons. In these cases of encephalitis the cerebrospinal fluid is usually clear and sterile, and the cellular count of the spinal fluid is usually negative as far as any particular findings are concerned. Encephalitis not infrequently ends in recovery with a residual paralysis.

Then, in addition to this, we have the cerebral type or the superior type of poliomyelitis, which you know occurs so commonly in July, August, September, and October, or until after the frost has come; and while it usually presents itself as a manifestation of a spinal lesion, it may occur with very marked meningocerebral symptoms, and in this way be confusing as far as the differential diagnosis is concerned. Of course, the epidemic nature of the disease, the very marked and diffuse muscular paralysis, and complete absence of reflexes make the differentiation easy at times.

Bear in mind that frequently there may be a fallacy in the examination of the cerebrospinal fluid. I want to call your attention to a case that was admitted several days ago where it was only on the third examination of the cerebrospinal fluid that the meningococcus was found; the two previous examinations of the cerebrospinal fluid were clear, free from cells, and contained no organism, and it was the third examination that showed a polynuclear leukocytosis and meningococci, and the baby then received the meningococcus serum and recovered, though it seemed very sick for many days. My point is, that even in cases of cerebrospinal meningitis the fluid may remain clear up to a certain time, and then it may become turbid and contain the meningococcus.

Concerning the prognosis in tuberculous meningitis there is little to say. Death is absolutely the rule and recovery is the exception. But, on the other hand, cases have been reported where there has been positive recovery from tuberculous meningitis. Recovery does result, but it is the exception. As to treatment, we have very little to say, as we have little hope from any known treatment at the present time. All we can do in these cases is to make the patients as comfortable as possible, treat them as mercifully as possible; give them all the aid and comfort that is in our power, and treat the symptoms as they arise. That is all.

TUBERCULOUS MENINGITIS. A DETAILED RÉSUMÉ OF THE CARDINAL SYMPTOMS. DIFFERENTIAL DIAGNOSIS. DESCRIPTION OF THE PERMANGANATE TEST OF THE SPINAL FLUID

How many recall this patient that I bring before you this morning? This is the little girl, Doris, that we showed to you five days ago. You will see at a glance she has not improved. Quite to the contrary, she has become worse. She is in coma. She resents being handled. The pupils are now irregular, the right being larger than the left; in the left eye she has a very marked conjunctivitis, and in the right you notice the desquamation of epithelium from the surface of the conjunctiva. It is collected in little masses and is abundant. Then note, too, the pallor of the face and the blushing or flush over the cheek-bones. You will observe there is a marked triangular pallor that begins at the root of the nose and comes down to the chin. The tongue is heavily coated and dry; notice the restlessness and the involuntary and incoördinate movements of her whole body.

Dr. Abt (to Patient): Doris, Doris, Doris. She does not answer when she is spoken to, pays no attention to her surroundings. She lies in the same position that we noted last week, on her side with her legs flexed on her thighs, and her thighs

very slightly drawn up. The knee reflexes are absent; the Babinski sign is present on both sides. The abdomen is sunken and the child has lost markedly in weight; the elasticity of the skin is diminished, although not so markedly as in some cases. Observe how she objects to being touched, even when the pulse is being taken. The pulse is moderately rapid and not particularly irregular. Yesterday's record shows the pulse-rate 112, 119, 124, 128, and 128. This morning the pulse is 92, as counted by the nurse. On the day previous, however, the pulse was slow, inhibited in character, and irregular, 92 one time, 84 another, 116, 84, 120, and 88. The spinal fluid has corroborated the findings. Let me again call attention to some of the points in the symptomatology of this disease.

The intelligence: With the onset of meningitis a mental hebetude is noted. These children are, as I have already said, dull; their mental perception is clouded, they have a great inclination to sleep, and occasionally the onset is marked by eclamptic attacks. The disturbances of intelligence increase as the disease progresses, and finally coma supervenes. Now this coma develops gradually: first, the patient is dull, then drowsy, soporous, and eventually comatose. This coma may occur in the last week of the disease, or it may occur a week or two before the end comes. Occasionally one sees a case where intelligence, more or less complete, is retained to the last. I have had children both here in the hospital and in my private practice where moments of intelligence occurred, and this return of consciousness was received with great glee by the parents of the suffering child. One should not be misled by such temporary return of consciousness, because every clinician has learned that this may be of a very temporary character; the patient soon relapses into coma and this continues until the end.

The convulsions: I have already stated that the disease may be ushered in by convulsions, and these are frequently Jacksonian in type, namely, half of the body is involved in the seizure. The convulsions may be tonic or clonic, or they may be both tonic and clonic. One must assume that the convulsions begin somewhere in the cerebral cortex on account of this Jack-

sonian type, but, on the other hand, the convulsions may bear some relation to the hydrocephalus. Hydrocephalus in these cases is marked and of a very high degree and causes some pressure on cortical areas, or it may cause a general increase of the intracranial pressure.

The tremor: Fibrillary twitching may be associated with those cases where convulsions have occurred or are occurring. As a rule, this occurs during the last week or ten days of the disease. The occurrence of convulsions very often marks the beginning of the terminal stage of the disease.

Paralysis may occur in these cases of tuberculous meningitis and may be observed early in the form of hemiplegia. Recently, in one of the foreign journals, Dr. Chancellor, of Chicago, described some cases of tuberculous meningitis characterized by hemiplegic seizures at the onset.

The rigidity of the neck is a very prominent symptom and may occur in three degrees—severe, medium, or moderate. Late in the disease the rigidity may be almost absent on account of a flaccid paralysis of the neck muscles. Rigidity is present in the majority of cases and continues until the end.

Kernig's sign occurs even more frequently, according to some recent statistics, than rigidity of the neck. It is usually bilateral, and, like the rigidity of the neck, in some cases is very, very marked, and in other cases it is only indicated or slightly present.

The Brudzinski sign is elicited, as I have told you, by placing one hand under the head and the other hand over the sternum and gently raising the head. If positive, the lower extremities are suddenly pulled upward and sometimes the toes describe the typical position of the Babinski sign, the large toe ascends and the other toes spread, fan-like.

Then the tache cérébrale is very frequently present. Indeed, it is one of the most common signs that we note in tuberculous meningitis. It may be said to be the most frequent, the most constantly present of all symptoms. This is a vasomotor affair and is elicited by stroking the body with the finger, the finger-nail, or with a moderately sharp-pointed instrument, and as a result there is an outlying zone of redness that marks

the point of stroking. Tache cérébrale occurs in a large number of febrile conditions other than meningitis.

In addition to this tache cérébrale we note very often a change in the color, *i. e.*, very frequently flushing alternates with marked pallor. The flushing is frequently observed over the cheek-bones. You will remember that we called attention to a peculiar triangular pallor in the case which we showed this morning. This condition is also seen in scarlet fever. Then, too, we notice a very great flushing of the body, particularly the side the baby lies upon, due probably to the same vasomotor disturbance.

We very often see facial paralysis, almost always unilateral, and the part most affected is the region of the mouth. Very rarely a double facial paralysis is observed.

In addition, an oculomotor paralysis is observed. The muscles of deglutition are frequently involved. Very often the nurse tells you at the height of the disease that the patient can no longer swallow. You ask her, "Are you keeping up the feeding, are you keeping up the nourishment?" and she replies, "I have tried, but the patient is unable to take food; the food regurgitates through the nose and the patient strangles." She says it is useless. If you inspect the mouth you very frequently see that there is a paralysis of the palate or of the pharyngeal muscles. This indicates an involvement of the glossopharyngeal nerve. There may be marked paralysis of the extremities, particularly in the form of hemiplegia, or there may be a paraplegia during the height of the disease; or in other cases the muscles of the trunk may be involved so that it is difficult or impossible for the patient to be placed in a sitting position.

The presence or absence of reflexes is variable. Sometimes the reflexes are markedly exaggerated; at other times they are diminished. It is generally agreed that the Babinski is of little value in young infants, as it may be a normal finding. Foot-clonus is frequently observed, especially in the spastic cases.

On the basis of the symptomatology, the disease is frequently divided into three stages: First, the stage of cerebral irritation, characterized by rapid pulse; second, the stage of cerebral inhibition, characterized by the slow or irregular pulse; third, the

stage of paralysis, or the terminal stage, characterized by tachycardia or very rapid pulse. It has been suggested that in the first stage the pulse has been accelerated on account of fever, but that is not true, because the temperature may be very low and yet the pulse be rapid.

The second stage, which is characterized by slow and irregular pulse, may change its character if sufficient atropin is administered over a certain definite period of time so as to cause a slight paralysis of the nerve-ends in the heart muscle. Thereupon the pulse is no longer slow, inhibitive, and irregular. One would suspect from this that cardiac irregularity was in part due to some pathologic change in the nerve-endings in the cardiac muscle.

The loss in weight is very marked. Most of these children tend to emaciate; it is a progressive loss. When a patient is attacked with tuberculous meningitis, he may be apparently robust, may be in a normal condition of nutrition. But, as a rule, the patient who falls ill with tuberculous meningitis is already much reduced in weight, health, and strength. This little girl here gives the impression that she has lost greatly in weight from day to day, and she is much emaciated compared with what she was at the last clinic day.

The Glands: Small lymph-glands are common in these cases, and may or may not indicate lymphatic involvement. Often a patient will have a large lymph-gland, but this may or may not play an etiologic rôle in the production of the tuberculous meningitis.

So far as the lungs are concerned, in a certain proportion of the cases there is an active tubercular process. As a rule, the pulmonary findings on physical examination are minimal. In a good many cases one finds simply the evidence of an acute bronchitis. It is true that this may be an acute tubercular bronchitis.

The temperature is variable in tuberculous meningitis. Toward the end or at the very termination of the disease the temperature may be very, very high, even 107° or 110° F.

Sometimes the breathing is extremely rapid or superficial. Most of us have seen these cases; and, indeed, this may occur

very early in the disease, two weeks or ten days before the end, and we think of an acid intoxication. In fact, many a clinician has hovered between a diagnosis of tuberculous meningitis and an acidosis. We often find acetone in the urine, but this is an elusive thing, and if one were to base his diagnosis alone on the presence or absence of acetone, he would often fall into error. So this superficial type of breathing, which is indicative of acidosis, should not confuse one. The Cheyne-Stokes type of breathing is present in many cases toward the close of the disease.

The abdomen is usually boat shaped, sunken, and this retraction is variously supposed to be due to paralysis of the abdominal musculature; but then we know that it is associated with great emaciation of the baby's body, with loss of muscular tonicity. In many cases the baby is in a starving condition on account of its inability to swallow; but, on the other hand, the child's abdomen may be distended, as it often is, and it has been frequently asked what the cause of this distention is. It was thought to be due to degeneration of the splanchnic nerves and consequent dilatation of the intestinal tract. It has been thought that it possibly was due to the swallowing of air. But we must explain the abdominal condition on the basis of some nerve lesion which may be present in the abdomen, possibly in some instances in the ganglia contained in the intestinal wall.

The enlarged liver so frequently seen is characteristic of miliary tuberculosis.

Let me say in closing that the eyes are involved in many ways. Conjunctivitis is frequent, as in this case, and one often observes little fields of epithelial desquamation. Keratitis is occasionally seen, and now and then an oculist finds a choked disk, optic atrophy, or choroid tubercles. The pupils offer a valuable sign in the diagnosis of the disease. They are usually dilated at the onset; the reaction may be slow; the pupils are often irregular, occasionally they are pear shaped; and very frequently there are changes in the size of the palpebral fissures.

It is necessary at times to differentiate between tuberculous meningitis and other diseases which resemble it. For example,

encephalitis sometimes presents the greatest difficulties, appearing with all the meningocerebral symptoms that we are accustomed to find in true meningitis—high fever, more or less unconsciousness, and paralytic symptoms. In some cases of encephalitis it is not uncommon to find paralytic phenomena rather early. In encephalitis of the superior type hemiplegias are extremely common; while in encephalitis of the inferior type it is not uncommon to find crossed paralysis, or lesions involving the pons. In encephalitis the cerebrospinal fluid is sterile and the cellular count of encephalitis does not vary materially from the normal. Many cases of supposed tuberculous meningitis which recovered were undoubtedly encephalitis.

You will recall, too, that during the season when anterior poliomyelitis is more or less epidemic, some cases present themselves as the bulbar type of the disease, and at the outset and, indeed, for several days a poliomyelitis may so closely resemble a tuberculous meningitis that the differentiation becomes difficult. Of course, the epidemic nature of the disease and the seasonal occurrence, the very marked and diffuse muscular paralysis, the complete absence of reflexes, and the negative spinal fluid findings make the differentiation fairly easy as a rule.

Tumors of the brain may cause difficulty in differential diagnosis. The tumor may be a solitary tubercle or an abscess, or one of the common forms of cerebral tumor. The examination of the spinal fluid, more particularly the cellular count and the chemical tests, throws the greatest light on the differential diagnosis.

Another word about the spinal fluid in tuberculous meningitis. The fluid usually flows out of the cannula under increased pressure. It is nearly always clear and colorless. In some cases, if the test-tube is held up to a bright sunlight, some fine flocculi are observed. If the fluid is allowed to stand in an incubator for twelve hours a thin coagulum forms which dips down into the fluid, and in this film the bacilli are contained. Some of this film should be obtained with a platinum loop, thinly spread on a slide, and stained with carbolfuchsin. As a rule, the bacilli are not numerous. In the hands of a careful technician

positive results will be frequently obtained. The globulin tests have been referred to. Lymphocytes, in the majority of cases, preponderate and are markedly increased in number.

I wish to add a word about making the spinal puncture. Make your puncture, preferably, in the fourth lumbar interspace, which is found at the level of a line drawn through the highest points of the iliac crests. Use a Quincke needle or cannula; avoid one with an excessively sharp point. Have the patient in the recumbent position on the left side. The patient should be brought well to the edge of the bed, while an assistant flexes the spine. The puncture is made in the midline and along the upper border of the lower spinous process. The needle enters in almost a horizontal direction. If the needle is thrust in too deeply into the canal the sharp point encounters a venous plexus situated on the anterior wall of the spinal canal. The admixture of the fluid with blood is objectionable because it interferes with the macroscopic examination of the fluid and makes the chemical examination of doubtful value. The puncture may be made while the patient is in the sitting position, particularly if the fluid is difficult to obtain. A specially improvised manometer may be attached to the cannula to measure the pressure of the spinal fluid. Occasionally the puncture results in a dry tap. If the fluid does not flow, another needle should be inserted above. Some sterile salt solution should be injected into the upper needle. If it escapes from the lower needle the conclusion is justified that both needles are in the subdural space.

THE PERMANGANATE TEST (demonstrated by Dr. A. Levinson)

The Permanganate Test is used for the purpose of determining the amount of organic substance in the spinal fluid. This test, which was originally described by Kubel-Tiemann for the determination of organic matter in water, has been employed for this purpose all over the world. Recently Dr. Mayerhofer, of Vienna, applied the test to spinal fluid. In so doing he found that spinal fluid in meningitis contains much more organic substance than the normal fluid. He also found that in certain diseases, like tuberculous meningitis, the different por-

tions of the spinal fluid contain different amounts of organic substances. This finding is utilized in making a differential diagnosis of meningitis.

The method of procedure in making a permanganate test is as follows:

The spinal fluid is withdrawn from the body into three tubes. The first tube should contain 2 or 3 c.c. of clear fluid (no blood); the second and third tubes should contain 10 c.c. each.

And while I am on the subject of the different portions of the fluid, it may also be well to point out the importance of this factor not only for the permanganate test, but for the determination of the cell count. It is a well-known fact that the first two or three cubic centimeters of the spinal fluid contain many more cells than the subsequent portions. This must be borne in mind in making a cell count if one wishes to avoid the danger of arriving at false conclusions, as the examination in one case of the first cubic centimeter and in another of the tenth, let us say, is likely to result in a great disparity; the latter normally will contain fewer cells and much less organic substance than the former. It is best to make all tests on spinal fluid, especially the permanganate test and the cell count, immediately after withdrawing the fluid from the body, for the cells as well as all other organic substances disintegrate quickly. To facilitate the cell count I should suggest the use of a preserving solution which will keep the cells intact for some time and also give a better view of the leukocytes. I have found the following solution very satisfactory: Methyl-violet, 0.1 gm.; glacial acetic, 2 c.c.; aquæ, q. s. 50 c.c. This diluent is drawn up into the pipet (white blood-cell pipet) as far as mark 1 and the spinal fluid as far as mark 11. Needless to say that this staining fluid cannot be used in determining the organic substances of the spinal fluid.

To return to the permanganate test:

Draw up 1 c.c. of the spinal fluid into a graduated pipet and empty it into an Erlenmeyer flask. As this is a chemical test, the vessels do not need to be sterile, but they must be perfectly clean. To the 1 c.c. of fluid add 10 c.c. of dilute sul-

phuric acid, diluted in the proportion of 1 to 3 (1 part sulphuric acid and 3 parts water). The sulphuric acid is used for the purpose of dehydration. You must dehydrate the substance or the permanganate will not oxidize well. Dilute the sulphuric acid and spinal fluid with 50 c.c. of distilled water; allow the mixture to come to the boiling-point and add to it 10 c.c. of decinormal permanganate solution. The permanganate solution can be bought or it can easily be standardized.

Our burette read 17.2; now you see it reads 27.2. After the solution is allowed to boil for ten minutes you will notice that the spinal fluid in the mixture has changed color—it is not a bright red permanganate any more, but a sort of dirty yellow, which means that the organic substances are reduced or oxidized. The color varies according to the amount of organic substance in the fluid. If there is a great deal of organic matter, the color will be a dirty yellow; if there is very little, the color of the permanganate remains unchanged.

At the end of the ten minutes 10 c.c. of normal oxalic is added. If you look at my burette you will see that it now registers 9.8. I pour in the oxalic to the 19.8 mark. When the 10 c.c. of normal oxalic is added the solution turns clear, the oxalic acid simply neutralizing the permanaganate. Of course, the permanganate and oxalic should be of the same strength. They should be standardized against each other, so that you know that 10 c.c. of oxalic equals 10 c.c. of permanganate.

My next step is to titrate the solution in the flask drop by drop until I obtain a faint red color. You see when a drop comes in it is red, but instantly it becomes neutralized and turns white. Eventually, however, the solution is titrated to a permanent red, after which the color does not change any more. We have reached that point now.

Next we ascertain how many tenths of a cubic centimeter of permanganate have been used up in the titration, and we take this amount as the oxidizing index. For instance, here we have 32.7 and we started out with 27.2. The amount of permanganate used up is 5.5 c.c. We therefore see that this spinal fluid required 5.5. c.c. of permanganate, or, in other words, the reduc-

tion index is 5.5. One should always make a blank test to find out whether the sulphuric acid by itself will not be reduced by the permanganate. In this case I found the required amount of permanganate for the sulphuric to be 1.2 c.c. The index, therefore, after I subtract the amount of permanganate used for the water and sulphuric acid, will be 4.3. The normal spinal fluid, that is, fluid from a patient not affected with any meningeal trouble, or even fluid from a case of meningismus (where there is irritation but no infection of the meninges, such as occurs in pneumonia or other infectious diseases), shows a reduction index of less than 2, sometimes even as low as 0.5 c.c.; 2 c.c. is taken as a standard for meningitis. Anything below 2 points to a meningismus or to a normal condition. Anything above 2 indicates a meningitis, probably a tuberculous meningitis. As the test of this fluid gave us a reduction index of 4.3, we may conclude that this is a case of meningitis.

In examining the three different specimens in this case we found that the different portions of the spinal fluid varied in the amount of organic substance which they contained, and that the first portion of the spinal fluid gave a higher index than the second portion, the second a higher index than the third. This is included in our diagnosis. A high permanganate index, especially when the first portion is higher than the second, makes the diagnosis of tuberculous meningitis reasonably certain.

From these indices we conclude that this is a case of tuberculous meningitis because a normal fluid usually gives the reverse—the first portion necessitating less permanganate than the second, and the second less than the third. A case of meningismus would also give a lower index in the first portion than in the subsequent portions. It is a meningitis only, and especially a tuberculous meningitis, that gives an index above 2, and a higher index in the first portion than in the subsequent portions.

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Professor of Dermatology in the University of Illinois

X-RAYS AND EPITHELIOMA

x-Rays have been used extensively for therapeutic purposes now for nearly fifteen years. The estimate of the value of the agent has been varied. There was first the period of cautious, tentative use; then the period of extravagant exploitation; and this was followed by a considerable period in which it lost ground in estimation. In the last two or three years it has been coming back. This revival of its use has been marked, and now, I believe, it is used more widely than ever before as a therapeutic measure. This recuperative power, so to speak, of radiotherapy —its ability to come back—is significant evidence of its actual value. Many remedies have a temporary vogue and then disappear, but in therapeutics, as in sports, resiliency is an evidence of inherent strength.

What are the actual facts in regard to the therapeutic value of *x*-rays? The time is too short to take up the whole subject, and I will confine myself to consideration of this question as regards my experience in the use of the agent in cutaneous epithelioma, in which it has had, perhaps, as extensive an application as in any other one condition.

Long experience has demonstrated that carcinoma tissue in the skin, which can be directly exposed, can be destroyed by *x*-rays. The explanation lies in the fact that carcinoma cells, like many other pathologic cells, are less resistant to the destructive effects of *x*-rays than are normal cells, and they can be destroyed by a bombardment of *x*-rays which is not sufficient to destroy the stroma of normal tissue in which the carcinoma cells are growing. In my opinion there is no possible ground for

denying this fact. With an experienced operator, the result can be produced with regularity. I am speaking now of carcinoma cells on the surface; not those protected by an overlying layer of healthy tissue. Accordingly, it is my experience that epitheliomas on the surface can be destroyed with practically unvarying certainty, whether they are basal cell carcinomas—rodent ulcers—or squamous cell carcinomas. Rodent ulcers—basal cell carcinomas—are likely to be more superficial, very rarely have metastases, and are more sensitive to *x*-rays; and as a rule their treatment with *x*-rays is easier. It is sometimes said that squamous cell epitheliomas will not yield to *x*-rays; that, in fact, their growth is apt to be stimulated by exposure to *x*-rays. This is decidedly not my experience. In my experience, while the ordinary squamous cell epitheliomas are somewhat more resistent to *x*-rays, they can be as definitely destroyed by *x*-ray exposure as can rodent ulcers. It so happens that the first prickle cell epithelioma that I ever treated I have kept in touch with since. The original condition, the histologic structure of the lesion, and the final result are shown in Figs. 38, 39, and 40. The case was treated in May, 1901, and the man remains well after fourteen years without ever having had a recurrence.

It has further never been my experience to see a squamous cell epithelioma that I thought was stimulated to a more rapid growth by the use of *x*-rays.

It is, of course, within the possibilities that the irritation of an epithelioma short of producing any degenerative effect in it may produce a stimulation of its growth, just as similar ineffective treatment of the epithelioma by other methods may make the situation only worse. But I have seen no results of this sort, either in rodent ulcers or in squamous cell epitheliomas, that made me feel that this danger was great with *x*-rays. On the contrary, my experience has been such as to indicate that even where the treatment was ineffectual, the action of the *x*-rays was still likely to retard the growth of the tumor by destroying at least part of the cells and damaging the vitality of many more. In short, *x*-rays have a definite and destructive action upon cutaneous epitheliomas, and with the

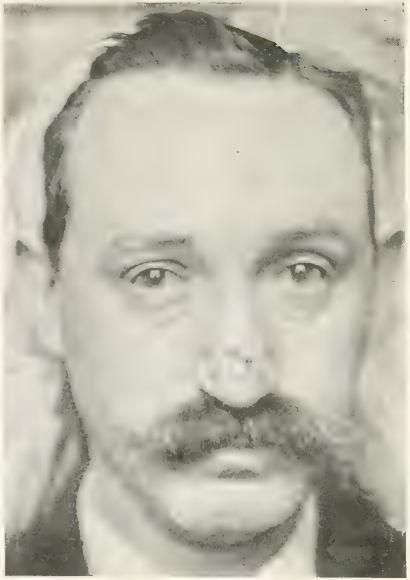

Fig. 38.—Squamous cell epithelioma of the nose. Treated with *x*-rays in May, 1901.

Fig. 39.—Photomicrograph from case shown in Fig. 38.

Fig. 40.—Result of *x*-ray treatment of lesion shown in Fig. 38. Photograph taken in March, 1912. Patient has remained well fourteen years.

experience that we have the agent can be utilized with assurance for the treatment of epitheliomas.

Of course, the successful treatment of epitheliomas with *x*-rays predicates a proper appreciation of the conditions involved in the treatment of epitheliomas, and a proper technic in the application of the agent. As for the selection of cases to be treated with *x*-rays, it requires the same sort of judgment that is required in deciding upon the operative treatment of epitheliomas. In one case the destruction of the lesion in the skin is all that is necessary. In another, the removal of adjacent glands may be required. In one case the problem may be simple because all of the epithelioma is easily accessible. In another case the prospect of cure may be lessened by the superficial or deep extent of the growth, by the involvement of bone, or by its extension into adjoining or underlying structures. In yet another, palliation is all that can be expected. These are the same factors that must be taken into account in the treatment of epitheliomas by operation; and with *x*-rays, as with operation, results are in considerable part controlled by experience and capable clinical judgment.

The rule which I have followed in determining whether operable epitheliomas were suitable for treatment with *x*-rays is the following: Wherever conservative surgery did not require the removal of deeper underlying structures or of adjacent glands for the operative treatment of an epithelioma, I have been willing to treat it with *x*-rays. In other cases, particularly in those where conservative surgery required the removal of adjacent glands, *x*-rays should not be substituted for operation. I formulated this rule, especially as regards the removal of adjacent glands, when I first began to use *x*-rays in treating epitheliomas. With it as a guide I have treated hundreds of cases, and I have never had occasion to feel that it was not a safe guide, or to reproach myself because I felt that any patient had not been given a proper chance of relief. *x*-Rays have proved in my experience an eminently safe way of treating suitable operable cases of epithelioma, and an agent which may give great benefit in many cases that are otherwise hopeless.

Where *x*-rays can be utilized, their advantages are many.

First, they avoid the necessity for operation.

Second, they avoid the destruction of unnecessary tissue.

Third, unless the operation is done with apparently unnecessary thoroughness, with correspondingly extensive destruction of normal tissue, I believe that *x*-rays actually give more radical results.

The avoidance of operation is, of course, an unessential advantage; an advantage of convenience. But, as a matter of fact, it has important practical advantages which I have had many physicians avail themselves of in their own persons. It means that many patients will submit to treatment, where otherwise they would postpone it or not accept it. It means also, as a rule, the avoidance of a stay in hospital, or of the suspension of the patient's vocation. In the case of feeble, old people, it means in many cases the escape from very real dangers which would be involved in operation. Operation has the advantage of being somewhat quicker in its results, but two or three weeks, with only an aggregate of a few hours actually given to treatment, will suffice for the cure of such epitheliomas as offer no difficulties of treatment by operation; and this length of time is not much in excess of that which is demanded for the cure of similar cases by operation.

The avoidance of excessive destruction of the tissues in which the epithelioma is situated is a great advantage for *x*-rays. In many cases this simply means the avoidance of scarring. With *x*-rays many epitheliomas can be gotten rid of with inappreciable scarring. In most successful cases the scarring is slight. In many extensive cases the resulting scar, in view of the extent of the lesion destroyed, is surprisingly slight. In certain locations this advantage is of great importance. This is particularly true about the nose and eyelids.

The radical operative treatment of many epitheliomas about the nose means the destruction of an ala, or the removal of so much of the tip or side of the nose that great and often disgusting deformity results. And this deformity cannot be gotten rid of, for a normal-looking nose cannot be made by any plastic

operation that involves the shifting of a flap. Many of these cases can be treated by *x*-rays with the production of no deformity whatever, and often with almost inappreciable scarring.

About the eyelids the advantage of *x*-rays over operation is of essential importance. A small epithelioma on the ciliary border of the lower lid, a frequent location for epitheliomas, is a serious matter for operative treatment, because radical operation requires the removal of part of the tarsal cartilage. This results in deformity of the lid, and almost certainly of deformity which will cause constant later trouble to the eye. Such cases can be treated with *x*-rays without any deformity whatever unless the epithelioma has itself already produced it. I have treated many such epitheliomas with *x*-rays or radium—which in my opinion does exactly the same thing—with completely satisfactory results. And yet within the year I have seen the statement that epitheliomas of the lid should never be treated in any other way than by operation. I am ready to maintain, on the other hand, that most epitheliomas of the lid should never be treated by operation unless it is impossible for the patients to avail themselves of treatment with *x*-rays.

The character of the results in epitheliomas treated with *x*-rays is sufficiently shown in the accompanying illustrations. The cases are selected with a view to illustrating different types of lesions and results. The length of time which has elapsed since these cases were treated shows the permanency of the results.

There are two essentials for the successful treatment with *x*-rays of cutaneous epitheliomas, as of other dermatoses. The first is, that the operator shall be familiar with the clinical conditions that he is attempting to treat, and the second, that he shall be familiar with his apparatus and its working. The great difficulty with the therapeutic use of *x*-rays in skin diseases has been that if the operator was familiar with the use of *x*-rays, he did not know skin diseases, or if he was familiar with skin diseases, he did not know how to use *x*-rays. The successful use of *x*-rays requires both a knowledge of the clinical conditions that are to be treated and a familiarity with *x*-ray application. The work cannot be done by rule of thumb. It must be con-

Fig. 41.—Epithelioma on the tip of the nose. Treated in March, 1903.

Fig. 42.— Result of treatment in Fig. 41. Patient remained well in August, 1913; ten years later.

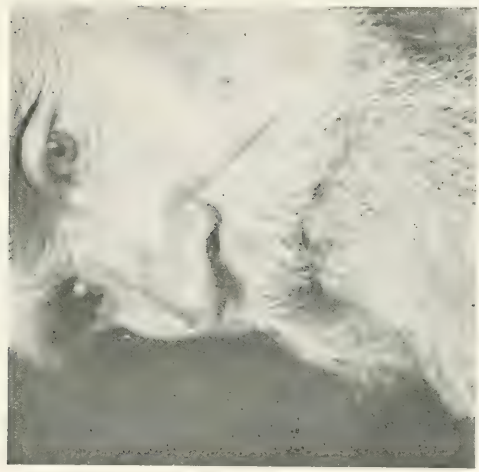

Fig. 44.—Result in case shown in Fig. 43. Died eight years later, without recurrence.

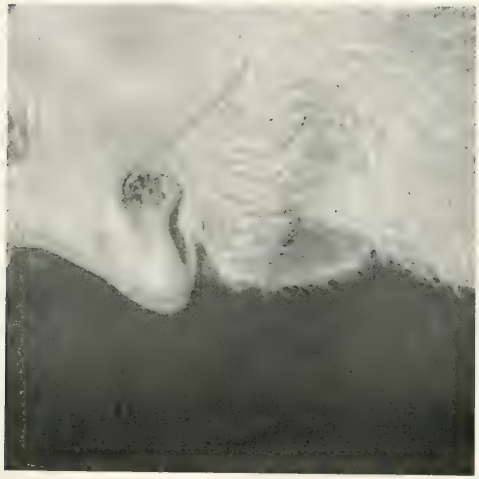

Fig. 43.—Epithelioma on ala nasi. Treated in October, 1902.

Fig. 45.—Rodent ulcer on cheek. Another one the size of a half-dollar, elevated, ulcerating on scalp. Treated in July, 1902.

Fig. 46.—Result in Fig. 45. There had been no recurrence in 1912; ten years later.

Fig. 47.—Epithelioma at the inner canthus of left eye. Treated in December, 1901.

Fig. 48.—Result in Fig. 47. Remained well six years later.

Fig. 50.—Result in Fig. 40. Remained well in 1912; nine years later.

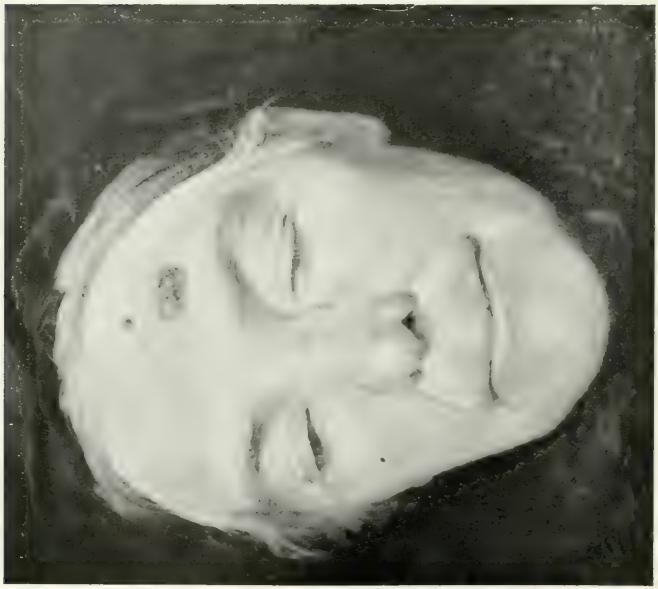

Fig. 40.—Nodular deep squamous cell epithelioma of forehead. Treated in April, 1903.

Fig. 51.—Large fungoid epithelioma in the skin at angle of jaw. Treated in July, 1903.

Fig. 52.—Result in Fig. 51. Patient died without recurrence six years later.

Fig. 53.—Squamous cell epithelioma on left temple. Treated in April, 1903.

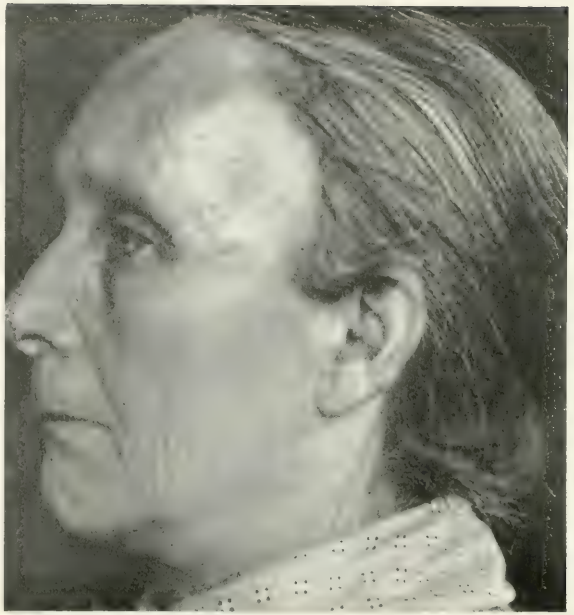

Fig. 54.—Result in Fig. 53. Epithelioma never recurred; patient died of acute illness October, 1908; five and one-half years later.

Fig. 55.—Very large ulcerated rodent ulcer on right temple. Treated April and May, 1913.

Fig. 56.—Result in Fig. 55. Patient remained well when last seen a few months ago; one and one-half years after treatment.

trolled by judgment and experience. It is particularly important for the successful use of *x*-rays for the operator to be so familiar with his apparatus and its working as to be able to produce a sufficient *x*-ray reaction without greatly overdoing it. If he has not this familiarity, he is likely not to proceed with the assurance necessary to get the proper effect.

In order to have this familiarity I believe it is highly desirable to develop a technic and to stick to it, with little variation. Some forms of apparatus are better than others, but the essential point in the successful use of *x*-rays is to have an apparatus with which you are familiar, and with which you know what you are going to do. I have found little reason to change my original technic, which was developed from the technic of Schiff and Freund. I use 10- to 14-inch coils; turbin interrupters, and regulating tubes, usually of 6-inch diameter. I am very cautious in the use of new tubes, and prefer tubes after they have been used and give off few short rays. For protecting surfaces not to be exposed I use lead masks $\frac{1}{50}$ inch thick. In the mask I cut a hole corresponding to the area which is to be exposed. Care should be taken that this hole is not unnecessarily large, but in the case of epithelioma it should extend well beyond the apparent border of the lesion.

In making the *x*-ray exposures there are two methods: One is to give weak exposures, and only after repeated exposures to get sufficient *x*-ray effect to get reaction—the broken-dose method, so to speak. The other method is to give one strong measured *x*-ray exposure sufficient to produce the reaction that is desired. My method is to give repeated weak exposures, and to continue them until the first appearance of reaction. I usually get a reaction which produces a first degree burn with vesiculation, and the subsequent formation of a necrotic pellicle. By giving such doses I get an *x*-ray effect which rapidly heals without discomfort. I could count on the fingers of one hand the cases in which there has occurred an *x*-ray burn that gave any serious pain or other trouble. My experience with the broken-dose method has been so satisfactory that I am, perhaps, prejudiced in its favor. I have at least a strong feeling that it gives the most satisfactory results.

CLINIC OF DR. FREDERICK TICE

Cook County Hospital

HEART DISEASE IN PREGNANCY. INDICATIONS FOR INDUCTION OF THERAPEUTIC ABORTION AND PREMATURE LABOR. TREATMENT TO PREVENT ARTIFICIAL TERMINATION. THE QUESTION OF MARRIAGE IN RELATION TO EACH TYPE OF CARDIOPATHY IN BOTH MALE AND FEMALE

This patient entered the hospital on the first of May. She She is thirty-seven years old, married, and gave this history when admitted: Her present complaint consists of shortness of breath, severe and unproductive cough, swelling of the feet and legs. The onset of her illness dates back to last year shortly after a confinement. Following her delivery there appeared shortness of breath and a severe cough. She is again pregnant, between the sixth and seventh months. The dyspnea and the cough have become relatively increased since this pregnancy. She complains of no pain or discomfort aside from shortness of breath.

This difficulty in breathing appears only on exertion, particularly when walking or going up and down stairs. When she is at rest the shortness of breath disappears. This dyspnea is associated with a cough which is usually unproductive; there is no history of spitting blood. Swelling of the feet has not been pronounced. There is no history of sore throat or of rheumatism. At the time of her last delivery, about one year ago, she suffered from a fever and the condition was diagnosed as puerperal fever. She was attended only by a midwife. A portion of the placenta was retained, according to her statement; the fever and chills appeared the day after labor and continued for six weeks. After

this she noticed a swelling of the feet, a dyspnea, and cough which has existed up to the present. The chills and fever continued until a curettage was done.

The family history: Father and mother both died in old age; seventeen brothers and sisters all dead, most of them dying in infancy. There is a history of having used alcohol some years ago in moderation. Venereal infection denied, both Neisserian and lues. Obstetric history: She has given birth to eleven children, the first child is thirteen, living and well, the second died at eight months of what was diagnosed as a meningitis. The third is living and well. All the other pregnancies have terminated either in abortion, premature delivery, or the child has died shortly after birth.

The record shows a temperature of 98° F., pulse 90, respiration 24 on the afternoon of the day she entered the hospital. The following afternoon the temperature was 99° F.; the following morning it was also 99° F.; in the afternoon 99.8° F. The next afternoon 100° F. The morning temperature, as a rule, is recorded as only 99° or 99° F. and a fraction, and the afternoon temperature reaches 100° F. or a little above. This condition continued to the 19th, and that morning the temperature was 101° F. and in the afternoon it was normal. Since that time the temperature has not exceeded 99° F.

The examination of this patient, particularly in reference to the cardiac condition, aside from the rate of the pulse, as already noted, reveals no abnormal or violent pulsation in the region of the heart, in the neck, or in the epigastrium. On inspection it is rather difficult accurately to locate the apex-beat.

On palpation, the most definite pulsation is in the fifth interspace in the nipple line. The apex-beat is slow and deliberate, the force, if anything, is relatively increased and quick in character. No thrill is present over the apex or over the base of the heart.

On percussion, the hepatic dulness is at the fifth interspace, the right border of the heart is one finger to the right of the sternum, the base of the heart at the lower edge of the second rib, the left border is just outside the nipple line.

On auscultation at the apex, there is a very definite presystolic murmur, beginning approximately in the middle of diastole, increasing and terminating suddenly in the first sound, which is loud and accentuated. No systolic murmur can be heard. The first and second tones are present. No murmur in the axilla. The second pulmonic is accentuated. No murmur over the base of the heart. The pulse is small, regular, and the tension is well sustained. A tracing has been made of this pulse; the lower tracing is the radial, and you can see there is not a sudden, abrupt elevation, but rather a long slow rise with a rounded apex; perhaps here accentuated because the tracing with the polygraph is made with the rapid speed.

The pulmonary examination reveals diffuse râles, more particularly behind and below, increased especially during the attacks of dyspnea, with the clinical manifestations of a pulmonary edema. The upper border of the liver is at the fifth interspace, the lower border not palpable below the costal margin, and the hepatic dulness not extending below this point. Positive findings in the abdomen of a pregnancy approximately in the seventh month.

We have, then, so far as the history and the physical examination are concerned, a cardiac involvement with the typical cardiac signs of a mitral stenosis. The presystolic murmur with the accentuated first sound, the small, well-sustained pulse—a pure, uncomplicated mitral stenosis. Associated with this a pregnancy of seven months. There are some points of considerable importance connected with this condition. The laboratory findings reveal practically nothing in the urine—a slight amount of albumin, a few casts. The white count is 8700, the blood-pressure, systolic, is 160. In searching for an etiology you will recall that this patient absolutely denies a previous throat infection, a tonsillitis, or a rheumatism; denies practically any previous illness; admits only her pregnancies and labors, and then, at the last delivery, an infection pronounced as sepsis. You will also recall that this patient has given birth to several children—the first and third still living, the second died of meningitis; the other pregnancies have all terminated in abortions, prema-

ture labor, or if the pregnancy went to full term the child died soon after birth. She denies a specific infection. A Wassermann on the blood has been done and the report comes back 2 plus. This we suspected and made a diagnosis of lues on the history alone, because we assumed that something had happened after the third labor. Why should a woman who has given birth to three normal children continuously abort, have premature labors, or children who die soon after birth? There is no outward evidence whatever of a specific infection, but the Wassermann gives a double plus.

This brings up the question, Is her mitral stenosis due to her luetic infection, or is it due to some other form of infection? It also raises the question of the matter of treatment. What should be done for this patient so far as her heart is concerned, and what should be done about the pregnancy? There seems to be a general belief that cardiac disease and pregnancy are incompatible. As a matter of fact, it is not an infrequent experience to have cases of pregnancy with heart lesions enter the hospital or the consulting room with the special request or demand that the pregnancy be terminated.

In reviewing the literature it is rather interesting to find that in some of the large obstetric clinics, particularly in Schauta's clinic in Vienna, in only one case in seven is the heart involvement detected. This probably is due to the fact that the examination is not so thorough when the patient enters for some definite disease or condition. It is also true that probably most of the cardiac lesions are detected in the obstetric department only when symptoms of disturbed compensation are present.

I can well recall an instance in this hospital, during my internship, when my colleague was called to the obstetric department to preside at a case then in labor, as the intern regularly in charge was out. In less than an hour the labor was completed, the mother was in excellent condition, the child living and well. When the regular intern returned a few hours later he was horror stricken to find this woman had been delivered. He threw up his hands and exclaimed, "That is the woman with the awful heart!" My colleague was very happy over the affair, blissfully

ignorant of the heart lesion, but the woman went through the labor without any trouble whatever.

In looking over the literature I should like to call your attention to the report of Fellner in the Schauta clinic in Vienna where 81 cases of cardiac involvement were systematically studied. In this number with mitral insufficiencies there were 26 that were compensated and 14 uncompensated. The mother died in only 1 of the uncompensated cases, and the child in the compensated 3 and the uncompensated 2. In mitral stenosis, in the compensated there were no cases, in the uncompensated 4; no deaths of the mothers, but 3 children died. In the double mitral, in the compensated 10, in the uncompensated 17 cases; 2 children died in the compensated and 10 in the uncompensated. In the aortic uncompensated there were 2, the mother died in 1 and the child in 1. Aortic insufficiency and mitral insufficiency, in the compensated there were 3, in the uncompensated 5. Of the children, 1 died in the compensated and 3 in the uncompensated. This gives a fair representation for the reason that the cases were very carefully studied. Since then Fellner has again carefully reviewed the records of some 900 additional cases with similar results.

Jaschke has made a report on 136 heart cases in 37,014 pregnancies, and his findings are almost identical with these. So from this you can arrive at practically this conclusion: in 81 cases with various forms of cardiac involvement, and even with the uncompensated lesions, the outcome so far as the mother is concerned is not unfavorable. That would agree very well with the observations and experiences we have had here in this hospital. A death during pregnancy or during labor, even in severe cardiac lesions, is rare and exceptional.

As to the outcome after delivery, that is an entirely different question. Rather frequently the condition becomes worse, the heart becomes more decompensated, and after a few months will go on to a fatal break.

This brings up, then, the question of cardiac disease in relation to marriage, pregnancy, labor, and the subsequent findings and occurrences in the puerperium. All authors and clinicians are

agreed that the individual with a heart lesion is just as likely to become pregnant as one without a heart lesion.

This is a question of great importance so far as the female is concerned, and must be considered not only on account of the heart condition, but of other contributing factors. Some authors say that marriage, even with pregnancy, has no great effect on the cardiac condition. This, however, is somewhat too extreme. It sometimes happens that a heart case does better after marriage than before, because the woman is relieved of the hard daily labor of providing for herself and can favor herself far more than when she was self-supporting.

As to the type of the lesion, it is quite true in certain forms of heart disease marriage is not advisable, nor is pregnancy or labor, and this is particularly true of mitral stenosis. In a mitral stenosis of high degree, especially where there is a history of symptoms caused by the lesion, marriage should be discouraged. In aortic insufficiency danger is relatively great, but aortic insufficiency is comparatively rare in the female. A patient with myocardial involvement with symptoms of disturbed compensation should be advised against marriage. In some of the arhythmias, particularly in the more serious types, marriage is not advisable. The same is true in some of the forms of heart-block, but it is also true that in some cases where the heart-block has persisted for years marriage has occurred with repeated pregnancies and labors without any bad results. Cases of angina pectoris are usually to be advised against matrimony.

So far as the marriage of the male with a heart involvement is concerned, the question is not so serious. Perhaps the greatest danger is associated with aortic insufficiency because at the time of intercourse the blood-pressure is materially raised, throwing a greater amount of strain on the aortic valves and left heart. If the lesion is associated with angina it should be considered a bar to marriage.

There is another very important problem associated with the male cardiopath, and that is the responsibility and increased amount of work necessary to provide for two; this is often materially increased with the advent of children, so that the work of

the wage-earner may be multiplied several times, and thus increase the strain on his heart. All these conditions must be considered and remembered in the cases of aortic insufficiency to determine the cause of the disturbed incompetency. In aortic insufficiency, in the absence of any other history, a luetic infection is always to be suspected, and it is always best to have a Wassermann. It is surprising how often the Wassermann is positive in the absence of any history or signs indicative of a luetic infection.

Now, as to the question of terminating the pregnancy in the presence of a cardiac lesion. How are we to know? Should all female cardiopaths who are pregnant be considered surgically and be aborted or have premature labor induced? By no means. It is probably safe to say that nearly all will go through pregnancy and labor without any greater danger than under ordinary conditions. How are we to know when the case should be terminated? In the first place, it is often very difficult to determine whether a heart lesion is present or not. The enlarged and distended abdomen interferes with the respiration, and the quite constantly accompanying secondary anemia makes the presence of murmurs frequent, and it is not an easy matter to determine when an organic lesion exists.

It is the opinion of Mackenzie, who has studied the question particularly, that a dilatation of the right heart with a tricuspid insufficiency in pregnancy is frequent; in fact, it is quite the rule during the labor-pains, coming and going, appearing and disappearing, according to the strain on the right heart. In the fatal cases of cardiac lesions, accompanied by pregnancy, the danger comes from pulmonary edema, but, clinically, our safest indication that the patient is approaching the danger-line is the appearance of dyspnea and cyanosis, and especially when these appear after exertion. When the patient on slight exertion becomes dyspneic or cyanotic, then the heart is easily disturbed and compensation is only being poorly maintained. When that happens, and more especially when edema appears in the ankles or lower extremities, and particularly when orthopnea occurs, there is great danger. The patient should then be placed in bed, just

the same as though no pregnancy existed, and given digitalis. If the heart becomes compensated by a reasonable rest in bed, the patient is allowed to be up and about, with a reasonable amount of exercise. Frequently she will go on through the pregnancy and labor without much trouble, but if there is a return of the symptoms, then the pregnancy should be terminated. This is particularly true if with a previous pregnancy dyspnea and cyanosis were also present.

So far as this patient is concerned, at the present time there is little or no disturbance of compensation; she is breathing freely and easily in a recumbent position. There is little or no edema, and while the urine showed on first examination a slight amount of albumin, it has now practically disappeared. There is no indication at present to interrupt this pregnancy. So far as we can determine, proper rest, digitalis, and the other measures that we institute in such heart lesions will be all that is necessary. There is nothing to indicate surgical interference.

When the conditions are such as to require a termination of the pregnancy, the rule is to institute the least surgical interference capable of initiating an emptying of the uterus by means as near physiologic as possible.

So far as the positive Wassermann is concerned, we have indications to institute at once vigorous antisyphilitic treatment. Whether this patient will go on to full term is a question; premature labor has occurred in previous pregnancies. During the last few nights she has suffered quite acutely from rather severe uterine pains, but there has been no hemorrhage and no dilatation. This condition may increase and she may go on and deliver at any time, and with that idea in view she will be transferred to the obstetric department for the possibility of an emergency delivery.

The antisyphilitic treatment will be instituted and pursued vigorously; her cardiac treatment will be carried on, but without any attempt at this time to interfere with the pregnancy.

(Patient taken out.)

One of the doctors has asked a question in reference to the patient just presented, and that is a question associated with the

matter of the history that she had a puerperal sepsis at the time of her last pregnancy. You will recall that the chart shows a slight afternoon temperature. The blood-cultures are negative and the explanation of this afternoon temperature is found in a slight exacerbation of the old valvular involvement. The autopsies on the fatal cases occurring after labor—as, for instance, where the death did not occur until two, three, or six months after delivery—almost always reveal the presence of a subacute endocarditis. The constant strain upon the heart incident to pregnancy and labor produces an exacerbation from which the patients are unable to recover, and they go on with a subacute endocarditis from which they eventually die.

PURPURA HÆMORRHAGICA. ETIOLOGIC FACTORS AND ANALYSIS OF THE PHENOMENA WHICH PRODUCE THE SYMPTOMS; ITS TREATMENT

DR. TICE: What is your diagnosis, Doctor?

INTERN: All I could find was an emphysema.

DR. TICE (to Second Intern): What is your diagnosis, Doctor?

SECOND INTERN: Emphysema. We could not get a history, as she speaks only Polish.

DR. TICE: Let me read the history to you: The patient entered the hospital on the 22d of May; she is fifty-nine, and through an interpreter it was ascertained that her present complaint consists of pains in her chest and stomach and of an eruption on her skin. She was well up to a few weeks ago, when she began to have pain in her left hand, and about a week later the left ankle was involved; on the same day bright red spots appeared on her left ankle, and a few days later they were also seen on her arms and on the back, evidently appearing on almost every part of her body. They were numerous, and it was observed that as some appeared, others disappeared, or

became dark and faded. Many of them became definitely blister-like in form and some of them hemorrhagic and actually bled. Vomiting came on, and this has persisted up to the present time; at first the vomitus consisted of a mucous, slimy material; later it became brownish. Vomiting ceased for a day, but returned. Since her illness she has been extremely exhausted. These attacks of vomiting are preceded by severe pain, located mostly in the epigastrium. The pain bears no relation to eating, nor is it relieved by taking food or alkalies. The pain was so severe that before she entered the hospital she had received hypodermic injections of morphin. She has had no chills or fever since she was admitted. She has been taking various medicines since the onset of her present illness; has eaten little or nothing because of the vomiting. She had typhoid five years ago. Has never had acute articular rheumatism or a similar disease. Her parents are both dead, cause unknown. She has nine children living and well; five other children born, but died at various ages, all within the first two years of life. Venereal history, according to her statement, is negative. She uses alcohol in moderation. Now, what is the diagnosis?

INTERN: It might be a purpura.

DR. TICE: What do you understand by a purpura?

INTERN: It is a hemorrhagic eruption and comes on sometimes during a rheumatic attack.

DR. TICE: Is it a clinical entity?

INTERN: Yes, it may be.

DR. TICE: A few points in the history: remember that the patient was seized first with pain in one ankle and then in the other, and then there appeared bright red spots over the surface of the ankles, and the following day they became numerous all over the body. Then, following this eruption, the presence of severe pains in the abdomen associated with nausea and vomiting. The eruption is still visible, but, unfortunately, not bright and typical like the early stage. However, you can observe over the upper extremities and to a slight extent over the lumbar region, but more particularly over the lower extremities, the evidences of this rash. At first bright red, gradually becom-

ing brownish; over these areas, where we have the crusts at the present time, there were slight vesicles, some of them containing hemorrhagic fluid, and over the larger areas a definite necrosis.

The physical examination of this patient reveals nothing, so far as the heart and lungs or the abdomen are concerned, aside from the existence of tenderness in the epigastrium. The urinary findings are: specific gravity 1015, no albumin or sugar, a few squamous cells. The white count 13,200; blood-cultures after forty-eight hours were sterile. The differential count is negative. Examination of the vomitus gave a very positive Weber. The stool (one examination only) was positive for blood. The sputum has not been examined. Cultures were made from the throat, even in the absence of any manifestation of a throat involvement, and they showed a few small bacilli and a few pneumococci.

From the history and from the condition which still persists in this patient, we can make the diagnosis of purpura. We would again ask the question, Is purpura a clinical entity? We can subdivide purpura into the symptomatic and the idiopathic or essential purpura. Of the symptomatic we will have relatively little to say at this time except to mention those due to an infectious, toxic, cachectic, neurotic, or mechanical etiology. These are symptomatic only, and the eruption is a manifestation of some other condition.

We approach now the essential purpura, and here again we encounter difficulties, for opinions vary as to whether we have a purpura which we can classify as a clinical entity. Particularly among the Germans it is quite the rule to classify all purpuras with hemorrhages under the one head of morbus maculosus, and under this term we have a large number of groups, as the purpura hæmorrhagica, the purpura abdominalis, the purpura urticans, and a number of others. It is also of interest, in the matter of the classification, to recall the opinion of W. Koch, that all the purpuras, including scurvy and hemophilia, are one and the same; that they are only clinical variations of the same process. Most of the authors are not of this opinion, and it would seem reasonable to place scurvy among the clinical entities, as well as

hemophilia. The question is, Are these one and the same? There is one type already referred to, the purpura abdominalis, also known as Henoch's purpura, that is characterized not only by the presence of the purpuric eruption, but by abdominal pain, with nausea and vomiting, with blood in the vomitus and usually in the stools. It may be associated with an acute nephritis.

This patient suffered from an arthritis, then the purpuric eruption, followed with pain in the abdomen, nausea and vomiting, and we can make a reasonable diagnosis here of purpura abdominalis. Now, as to the other manifestations: This patient has had no fever; that is quite the rule. She has a leukocytosis; the blood-cultures are negative; the throat cultures were negative except for one organism, presumably the pneumococcus. Reference has been made to rheumatism, but this patient does not give a history of rheumatism. She has, however, complained of arthritic disturbances during this disease. Was that a rheumatism or not? What relation does the purpura bear to rheumatism? Perhaps the belief is entertained that purpura and rheumatism are intimately associated, but it is often exactly the opposite. Practically all of these patients will give a negative history as to previous attacks of rheumatism. They have not had rheumatism, but an arthritis appears at about the time of the purpura. Then we know that in rheumatism the endocardium is involved in approximately 70 per cent. of the cases; this patient has no endocardial complication.

Then, again, if purpura is associated with or due to a rheumatic infection, why do we have so many cases of rheumatism without purpura? It is relatively infrequent as we recall the cases. So we can well conclude that there is an essential difference between a rheumatism and a purpura, or the ordinary clinical arthritic rheumatism.

What is the cause of purpura? The present belief is strongly in favor of an infection. That we have some type of infection is evident from the arthritis, the cutaneous manifestations, the abdominal disturbances, the leukocytosis, and the occasional positive blood-cultures. When we know more concerning the actual conditions which exist it probably will be

determined that these purpuras are really infective. At the present time a large number are grouped under the symptomatic, and others under the essential purpuras, but the essential purpuras will be more accurately classified until perhaps few, if any, remain. Today our terms practically mean nothing more than a clinical convenience.

So far as the eruption is concerned, its pathogenesis is not definitely known or understood, but presumably, in the cases examined up to this time, there occurs in the capillaries a hyaline degeneration which leads to thrombosis and then to a local hemorrhage; or possibly the condition results from the effects of the toxins upon the circulation, or an embolic process would account for these cases much better. If the condition is due to a toxin why should the eruption appear in crops, sometimes only one or perhaps two or three showers after a day or two? If the vessels are degenerated, then why should the condition not continue and the purpura extend over several months? It is hardly to be expected that the condition would disappear so soon if the vessels were degenerated.

If it is an embolic process we can understand how it occurs in crops. The cutaneous manifestation is visible; the eruption may be very small, the size of the point or head of a pin, and it is characterized clinically by the fact that the spots do not disappear on pressure. When the hemorrhage is large, it is known as an ecchymosis, and sometimes the eruption is definitely of a ring formation. A red ring may surround a white, anemic center, and these are the ones which go on to necrosis—a typical embolic process.

Now, as to the abdominal manifestation: Why do these patients have pain, with nausea and vomiting; frequently with hemorrhages from the stomach and bowels? Why do they suffer from an acute nephritis? It is likely that small or large hemorrhages involve the stomach, the intestines, and also the kidneys, due, probably, to an infection or the result of toxins. The gastro-intestinal and kidney manifestations have been explained on the occurrence of an internal urticaria. As you know, a cutaneous urticaria or erythema is frequently associated with a purpura,

and the same thing may occur in the stomach, intestines, and kidneys.

It is also interesting to call your attention to the fact that these cases are not infrequently operated. Laparotomies have been done because of the possibilities of some acute surgical disease, as a gall-stone, appendicitis, or gastric ulcer, and in one case reported in Boston urticarial wheals were present, involving the intestinal wall.

So far as the prognosis is concerned, it is relatively good, particularly in the mild cases. In the severe cases, with hemorrhages from the stomach and bowels, the prognosis is unfavorable.

The treatment consists in keeping the patient in bed, and where there is much tendency toward hemorrhage the administration of horse serum and calcium lactate to increase the coagulability of the blood. Where there is much pain in the joints the use of sodium salicylate or aspirin, and where the pain is very severe it may be necessary to use morphin or codein.

INFANTILISM—THE RÔLE OF THE INTERNAL SECRETIONS

DR. TICE: What are your findings in this case?

DOCTOR: A case of underdevelopment with a double mitral lesion.

DR. TICE: What is the diagnosis, aside from the heart?

DOCTOR: Infantilism.

DR. TICE: Infantilism, a term that is used to include a number of various groups. It was first used to indicate the physical and mental characteristics of childhood passing on into adult years, but since the coining of the word various groups have been picked out and described as pure clinical types. What relation does this cardiac condition bear to the general condition?

DOCTOR: I don't think it has any relation. She has had tonsillitis, although her heart has not given her any trouble.

DR. TICE: This patient entered the hospital on the 19th of April, with the following history: She is twenty-three years old; has never been healthy and robust so far as she can remember. The present illness began three weeks ago, when she had a cold in the head, unaccompanied by chills or fever. There was a general muscular soreness involving practically all the muscles of the body. This condition lasted for about a week, during which time she developed a bronchitis which was accompanied with a severe headache and pains in the eyes. Four days ago a definite pain appeared in her chest on the right side, which was not influenced by respiratory efforts. Later, pain appeared over her heart. She has had three separate and distinct attacks of rheumatism, and also measles in infancy. Her parents are living and well; has four brothers and three sisters living and well; two brothers and one sister died in infancy.

More particularly in reference to the pain in the small of her back and the right side of her chest: the onset was sudden and abrupt, like a knife stab, but it was uninfluenced by respiratory movements. She has had previous attacks of pain of similar character accompanying her rheumatism; these attacks lasted for a week or two and then disappeared. The patient has never menstruated. She has been married four years. There is an absolute lack of any sexual desire or pleasure. Denies the use of alcohol except in very small amounts; no history of any specific infection. I will have her stand so that you can get some idea of her height; she measures 4 feet 5 inches, and can easily stand under my arm, a very striking underdevelopment when we take her age into consideration. Her sisters and brothers are all of ordinary build; a sister fourteen years old is a full head taller. The father and mother are of usual height. Her husband is also underdeveloped; as yet we have not examined him, but are waiting for the opportunity. The patient presents a striking picture of underdevelopment or infantilism.

In the physical examination there is very little to which to call your attention. The underdevelopment is evident and striking;

another feature to which we must direct our attention is the presence or absence of the hair. So far as development and abundance of the hair on the scalp are concerned, that is of the usual type; the eyebrows and face present nothing out of the ordinary, but when we examine the axillary and pubic regions we note a complete absence of hair; there is no hair on the pubes or vulva, or only a few small rudimentary hairs, as you can observe, and, according to her statement, there has never been more than there is now. There is also a lack of development of the mammary glands, and you must not only remember her age, but also that she is married. The sexual function has not stimulated any glandular development. Examination of the thyroid reveals nothing abnormal, the gland is easily palpable, or at least there is a mass present which we interpret clinically as the thyroid. There is no increase or decrease of the normal dulness over the base of the heart. The liver and spleen are not palpable.

The physical examination of the heart reveals a slight increase in cardiac dulness to the left, with relatively a greater increase to the right, and on auscultation at the apex a presystolic-systolic murmur. The interpretation of the cardiac findings is comparatively simple—she has the ordinary double mitral lesion (mitral stenosis and mitral insufficiency).

Vaginal examination reveals an infantile uterus, no tubes or ovaries can be palpated.

Now, as to the condition of infantilism, for which she did not enter the hospital, that is an altogether different problem. As already noted, the term was originally used to designate the youthful qualities extending on into adult years. The question has already been asked, Does this heart bear any relation to the underdevelopment, or is the condition an independent one? If it is an independent one, then what is the cause of this underdevelopment? You have already heard that she has brothers and sisters who are perfectly normal in size; the same is true of her father and mother, but it is evident that, so far as her development is concerned, she is far below the average.

This is a question that has been extensively investigated during the last few years, particularly since the nature and function

of the internal secretions have attracted so much attention, and it is quite definitely determined at the present time that we have to do here with a disturbance of the internal secretions resulting in arrested development, as opposed to an overactivity resulting in gigantism or acromegaly or some other type of excessive growth. In this condition practically all of the discussions and investigations, more particularly those concerned with the French school, seem to hinge upon thyroid disturbance, and it is true that a considerable number of cases must be included in the group due to abnormal thyroid function. More recently clinical studies have determined that we have other internal secretions capable of producing identical or similar types, and these are especially concerned with the interstitial cells of Leydig in the testicles and the corresponding cells in the ovaries, also the secretion of the pituitary and the suprarenal glands. There is also a pancreatic type and an intestinal type. The Doctor will place on the blackboard the classification of infantilism by Anton, modified by Dietrich, based chiefly on the pathogenesis or etiology. There are various classifications, clinical and otherwise, but a clinical classification is hardly permissible or possible according to our present knowledge. The same criticism may be made as to etiology, but there are some points which seem to be in very little doubt at the present time.

The first group is, Infantilism without detectable cause. On account of inability to determine any definite reason it has been assumed that the underdevelopment results from defectiveness of the germinal cell.

The second group is, Infantilism due to the disturbance of the internal secretions. Under this we have some sub-groups: (*a*) With myxedema or cretinism (thyrogenic); (*b*) with absence or underdevelopment of the genitals (dysgenitalism); (*c*) infantilism due to disturbance of the various visceral glands, as the thymus, parathyroid, suprarenals, pancreas, etc.

Third, Infantilism due to other internal disturbances: (*a*) Primarily brain diseases; (*b*) angioplasia.

Fourth, Infantilism due to toxic or trophic disturbances: (*a*) Intoxications of the parents (alcohol, lead, mercury, etc.); (*b*)

congenital lues; (*c*) extra-uterine diseases, as tuberculosis, valvular diseases, pellagra, malaria, etc.

Fifth, Mongolism.

According to this tabulation an attempt has been made to classify from the pathogenesis: Infantilism associated with a disturbance of the germinal cell; infantilism resulting from disturbance of the internal secretions, as the thyroid; from the absence or underdevelopment of the genitals, the so-called dysgenitalism; infantilism due to the disturbance of the various internal glands, such as the thymus, the thyroid, the pancreas; infantilism due to internal disturbances, such as brain tumor; infantilism due to toxic or trophic disturbances, as may result from the use of alcohol (the underdeveloped child of the alcoholic is a familiar illustration) or in syphilitic infection, or the result of acute infectious diseases during pregnancy; and then the fifth group, Mongolism.

If we wish to classify the case before us we can possibly disregard the first group. The second group, infantilism associated with a myxedema and cretinism, we can also at once discard. Infantilism with underdevelopment of the genitals is very important. The disturbances due to defective secretion of the testicles and ovaries have been very well emphasized by Tandler and Gross, of Vienna. The custom of castration practised as a religious rite in some countries demonstrates the effect of deficient testicular secretion upon the development, producing, as you know, the condition of eunuchism. In these we have two separate and distinct groups, the one known as the long-legged eunuchoid, on account of an overdevelopment of the long bones. The epiphyseal growth is not completed at the usual time and these individuals sometimes reach the age of thirty-five or forty before the growth ceases. The other group, instead of showing an increase in height, is characterized by hyperdevelopment of adipose tissue, a high-pitched voice, and beardless face. So we have the two separate and distinct types—the definite overgrowth of the long bones and the obese—the latter is usually referred to as the Brissaud type.

In this group, in which the infantilism is due to a disturbance of the various visceral glands, it is well to include the pituitary,

for the reason that it is one of the internal glands that is very intimately associated with the function of the testicles and ovaries. In a pituitary disturbance the abnormality depends upon which lobe of the gland is affected, as has been so well demonstrated recently both experimentally and surgically. The anterior lobe is concerned with the growth of the individual.

If an increased activity occurs in it before growth is completed, gigantism occurs, but if normal growth has been attained, it results in acromegaly. An underdevelopment of this gland is concerned in some of these types of infantilism, and it is a very difficult problem to determine whether we have a separate clinical type or whether we have a dysgenitalism, or a combination.

It seems likely from what we can determine that a disturbance in one gland produces a disturbance in the others, as they are intimately associated and correlated. It has also been definitely determined that ossification of the long bones is concerned with the internal secretion of the testicles and ovaries and that overgrowth is concerned with disturbances in the pituitary. Skiagrams of the long bones of this patient show that the epiphyseal line is still demonstrable (Fig. 57). She is twenty-three and ossification is not complete. She is underdeveloped, the very opposite of what you might expect, and that would seem to demonstrate that the failure to ossify is attributable to disturbed ovarian function, and the failure of growth is possibly due to pituitary trouble.

There is one form of pituitary disturbance from defective secretion, characterized by adiposity and underdevelopment of the sexual glands, known as Fröhlich's type. There is another form of so-called dystrophic infantilism, known as the Lorain type, which is characterized clinically not by an overgrowth, but the individual maintains a relative proportion; that is, the arms and legs are in proportion to the body, but much underdeveloped. That condition evidently exists here.

In endeavoring to obtain more knowledge as to the probable etiology in this case some four or five skiagrams were taken of the head, particularly to show the sella turcica and to determine indirectly the size of the pituitary. As the gland increases in

size the sella turcica enlarges, and by its size we arrive at the approximate size of the pituitary. If the gland is diminished in size the sella turcica is correspondingly small, and that is what we see in these various figures.

Fig. 57.—x-Ray of hands. Note presence of epiphyseal line at lower end of radius and ulna; also in phalanges.

With a pituitary involvement, as you know, there is frequently an associated disturbance in the posterior lobe of the gland, which produces variations in the metabolism of carbohydrates and fats. In those cases where the secretion is increased there

is a diminution of the sugar tolerance, but when the activity is lowered there is an increase in the sugar tolerance. This patient was given 250 grams of glucose, but no sugar appeared in her urine; a few days later she received 300 grams, without any sugar in the urine; so we must conclude that her sugar tolerance is above normal.

Fig. 58.—*x*-Ray of skull. Note small size of sella turcica.

Particular interest is associated with this Lorain type of infantilism in its frequent association with a syphilitic infection. A Wassermann was made on this patient and the report was triple plus. There is no history that would lead us to suspect acquired syphilis. In the family history, however, there is the fact that two brothers and one sister died in infancy, the cause of

which was not known or determined. It is quite likely, with this history and the triple plus Wassermann, inherited lues is the cause of this infantilism.

As to the question of whether or not it is due to an interference with the function of the ovaries, the pituitary or the thyroid, and perhaps some of the other internal glands, or, more likely, a combination of disturbances, it is quite impossible to say.

The diagnosis consists in:

1. A double mitral lesion with perfect compensation. The endocardial involvement resulted, probably, from the recurring attacks of rheumatism.

2. Infantilism of the Lorain type. Etiologically due to a congenital lues producing a disturbance of the internal secretions.

As for the pain in the back and right side, no physical signs have been found to establish a diagnosis. Perhaps it was due to a pleurisy or may have been muscular.

Treatment: You will recall that this patient entered the hospital for the pain in her back and side; the cardiac involvement and infantilism are only incidental findings. As the pain has disappeared, no treatment is required. The heart is perfectly compensated, so cardiac drugs are contraindicated. The congenital lues, the probable etiologic factor in producing the disturbance of the internal secretions resulting in the infantilism, demands active antisyphilitic treatment. This alone may result in considerable improvement as soon as a more normal relation of the internal glands is established. Following or accompanying the antisyphilitic treatment extracts of some of the internal glands, as the thyroid, ovaries, etc., may be tried.

MARIE'S PULMONARY OSTEO-ARTHROPATHY: A SYNDROME FREQUENTLY ASSOCIATED WITH CHRONIC PULMONARY INFECTIONS

DR. TICE: What is the diagnosis?

INTERN: This is going to be a snap diagnosis because he would not talk and would not let us make a thorough examina-

tion. We found some râles behind and made a snap diagnosis of tuberculosis.

SECOND INTERN: I think there must be a cardiac involvement. He says his fingers have been that way for a long time.

DR. TICE: Yes, clubbed fingers or drum-stick fingers. This condition has been known ever since the days of Hippocrates and is known as the "Hippocratic fingers." The same characteristics exist in the feet; there is a definite clubbing, drum-stick formation of the toes. Did he tell you anything about his cough and the amount that he is expectorating?

DOCTOR: No, he wouldn't tell us anything.

DR. TICE: This (picking up sputum cup) is partially full, and is the second sputum cup he has had this morning; the first one was easily two-thirds full. He probably will expectorate a large amount before he leaves the clinic. It comes up in great quantities, following a paroxysm of coughing.

I will read the history to you: The patient is forty-three years of age, and entered the hospital on the 29th of March, complaining of a cough, with pain all over his body, night-sweats, and loss of weight and strength. Three and a half years ago he was operated in one of the hospitals of this city for an inguinal hernia, for which an ether anesthesia was given, and following the operation he suffered from an ether pneumonia of the left lower lobe. The pneumonia did not resolve, and because of the persistence of the temperature and sweats a diagnosis was made of empyema. Punctures, however, were always negative.

Three or four months after his operation he left the hospital and was admitted to one of the tuberculosis sanatoria in the vicinity of Chicago. There he remained for a time under observation; repeated sputum examinations were made, but were persistently negative for tubercle bacilli. He was finally discharged, with the statement that he had no tuberculosis. At about that time further sputum examinations were made at various laboratories in the city, and probably two or perhaps three dozen specimens were carefully examined, and all were negative. He was suffering from a persistent cough accompanied by a fair amount of expectoration, but with little or no temperature. He finally was

sent to this hospital about twenty-two months ago; was under observation for a couple of weeks without determining anything more than was known previously. He still had persistent signs of infiltration of the left lower lobe, with the sputum constantly negative. This continued up to three months ago, when he visited my office one afternoon and was in practically the same condition he is in now, but perhaps a little stronger, with a history of attacks of coughing and expectorating enormous quantities of sputum, at times definitely offensive—not the stinking odor of a gangrene, but more like that of decomposing cabbage.

The clubbing of the fingers and toes appeared shortly after he left the hospital following the operation for hernia, and has gradually increased up to the present time. It is chiefly for that reason that he is brought into the clinic this morning.

Would you designate the deformity of the hands and feet by any special term?

INTERN: It may accompany a heart disease.

DR. TICE: Is there any term by which we designate this condition? What is the trouble here with these hands and feet?

DOCTOR: They become enlarged in acromegaly.

DR. TICE: You can see at a glance that this is not an acromegaly. There is no change in the skull and the fingers are not typical of acromegaly. This is a definite drum-stick deformity, which does not occur in acromegaly.

DOCTOR: It might be an osteo-arthropathy.

DR. TICE: Yes, more definitely, Marie's hypertrophic pulmonary osteo-arthropathy, first described by Bamberger, of Vienna, in 1889, and later by Marie, in 1890, who published and illustrated several cases. It occurs chiefly in pulmonary disturbances. Alexander, in St. Bartholomew's Hospital Report, London, in reviewing 103 cases of this disease, found a chronic pulmonary involvement in 77 of the cases.

That brings up the question of the etiology and pathogenesis. Why does this condition occur? According to Marie, it results from an infection or intoxication. There is a toxic condition which produces a toxic arthritis, and the toxin invades both bone

and soft tissue. Some authors emphasize the existence of syphilis as a causative factor. Others advance the theory of tuberculosis, and so far as can be determined from Thornton, he would lead us to believe that it is a tuberculosis of the joint, although subsequently he points out that the tuberculosis may exist in the lungs and that the local condition is toxic only.

Then there is the neuropathic theory that ascribes it to a disturbance of the nerve supply to the part, particularly because in some instances there are definite sensory and trophic disturbances, a condition perhaps analogous to that formed in syringomyelia. Two years ago we had a patient here who presented a definite clubbing of the fingers of the right hand, on the same side of a subclavian aneurysm. In a case reported in the literature the clubbing disappeared after surgical interference for the aneurysm. The patient we had here was also operated, but left the hospital before we could determine whether the clubbing had disappeared or not. When associated with aneurysm it is presumably due to interference with nutrition to the part or pressure of the aneurysmal sac upon the nerves.

Now, as to the anatomic changes that occur. There are 16 recorded cases of this condition in which complete autopsy, including the osseous examination, reveals the typical anatomic conditions that we look for clinically, consisting of changes in the bony structures and also in the soft tissues. When we examine this hand and forearm we are struck not only with the clubbing of the distal end of the fingers, but with an apparent thickening of the hand and particularly a thickening of the wrist; a great increase in the transverse diameter of the wrist, an apparent increase clinically of the lower ends of the radius and the ulna. The upper portion of the bones of the wrist show no change. The same is also true of the lower extremity, the clubbing of the toes with an increase in the substance of the feet, but more particularly of the malleoli. There is no definite change in the upper portion of the forearms or the legs. We will show you a skiagram of the hand. Bear in mind that these anatomic studies reveal an increase in the osseous structure involving not the carpal, but the metacarpal bones, and especially the phalanges

and the lower end of the radius and the ulna. In this picture you can observe no change in the carpal bones, a slight change in the metacarpal, and more particularly an increase in the first group of the phalanges. The distal phalanges remain absolutely normal, with the exception of small osteophytes, but that is not to be interpreted as pathologic; that occurs under normal

Fig. 59.—Picture of second patient with pulmonary osteo-arthropathy, showing clubbed fingers.

conditions. Clinically, there is a clubbing of the fingers, but the skiagram reveals no increase in the bony substance. The clubbing is due to an increase in the subcutaneous deposit of fat.

A skiagram of the feet would reveal changes identical with those in the hands. I will show you later some pictures of another case presented here in this clinic, perhaps four years ago, showing a more typical picture of Marie's osteo-arthropathy.

When I saw this man in my office, about four months ago, the examination revealed a bilateral involvement of the lungs, with

Fig. 60.—*x*-Ray of hand. Note subperiosteal thickening, particularly of the first row of phalanges and lower end of bones of forearm.

the findings more pronounced over the left lower lobe. The sputum, which is expectorated in abundance after each parox-

ysm of coughing and has a very foul odor, revealed for the first time the presence of many tubercle bacilli, and that, you will recall, was after the lapse of two and one-half years that this man had been under observation. The skiagram reveals more findings on the left side. That shadow, with the suggestive clinical pic-

Fig. 61.—Feet of second patient, with clubbed toes.

ture, the paroxysms of coughing, expectoration of a large amount of foul-smelling material, at once must suggest a bronchiectasis. These symptoms do not suggest the presence of a pulmonary cavity, but are the clinical manifestations of bronchiectasis. That is the condition which occurs in approximately one-fourth of the cases of Marie's osteo-arthropathy.

We have here pictures of the other case referred to, presented at the clinic four years ago, in which we made the clinical diagnosis of pulmonary tuberculosis with a cavity. This cavity shows very beautifully in the apex of the right upper lobe. No

Fig. 62.—Second patient, with arthritis of knees and ankles.

tubercle bacilli were found in the sputum, but this patient, from the history and the physical examination, no doubt had a tuberculosis. The pictures are even more typical of an osteo-arthropathy than in this present patient. There is a hand showing

great thickening in the first row of phalanges; the same is true of the second group, and other pictures show a thickening in the ulna. The same condition is also shown in the feet; the involve-

Fig. 63.—Marie's hypertrophic pulmonary osteo-arthropathy. *x*-Ray of chest. Shadow increased on left side, especially below.

ment not of the tarsal, but of the metatarsal bones and the first and second rows of the phalanges. Clinically, the patient suffered from an arthritis. Both wrists, knees, and right hip were in-

volved. The left knee was aspirated and a considerable amount of fluid was removed; the fluid was examined carefully, guinea-pigs were inoculated, but the tests were all negative. The knees show but little change, while there is some periosteal thickening of the tibia and of the femur.

Fig. 64.—*x*-Ray of chest of second patient. Note shadow of cavity in right apex.

So we have from the clinical standpoint a typical illustration of Marie's hypertrophic pulmonary osteo-arthropathy, which is a syndrome, a condition associated with some other condition, and in most instances with a chronic pulmonary infection. It

results presumably from the influence of toxins upon the osseous structures as well as the soft tissues, producing the deformity.

Fig. 65.—*x*-Ray of hand. Note more or less subperiosteal thickening of all the bones, but especially those of the first row of phalanges.

It is conceivable that when our laboratory methods are further perfected it will be possible to find the bacilli, but so far this has not been accomplished.

Here is an old, persistent pneumonia, perhaps originally due to a pulmonary tuberculosis or some other type of a pulmonary infection, producing today a bronchiectasis, resulting in an osteo-arthropathy.

Fig. 66.—x-Ray of feet. Note subperiosteal thickening, especially of the metatarsal bones and those of the first row of phalanges.

As to the prognosis, that depends not upon the osteo-arthropathy, but upon the primary disease, and that, of course, is clearly unfavorable.

The treatment consists in an abundance of fresh air, nutritious food, and general tonics to build up the physical condition as far as possible. The treatment of bronchiectasis is best accomplished by the intratracheal injection of antiseptics. Perhaps one of the best means of controlling this offensive expectoration is by means of the creosote vapor baths. At first there is considerable irritation, but in a short time the patient becomes accustomed to the fumes, and after ten or fifteen minutes of this treatment will expectorate enormous amounts of sputum and will be relieved for thirty-six or forty-eight hours, when the treatment can be repeated.

In this and other cases we have tried the administration of autogenous vaccines and stock vaccines, but without any improvement whatever. We have also tried the administration of urotropin, but the condition is merely alleviated, and will go on to a fatal termination.

CLINIC OF DR. WALTER W. HAMBURGER

COOK COUNTY HOSPITAL

CARDIAC NEUROSIS. PSYCHIC EFFECT OF HEART DISEASE ON PATIENT. NECESSITY OF TREATING THE INDIVIDUAL AS WELL AS THE DISEASE

THE patient I wish to present to you this morning is a tailor, twenty-eight years of age, who has been sick for three weeks. His case brings out some of the points that we have been discussing and, although he is not a hospital patient, he has consented to come up here to the clinic this morning. I saw him for the first time at his home on Thursday, a few days ago. Will you (addressing Patient) tell these doctors about your complaint?

PATIENT: I wanted to join a lodge two months ago, and the doctor who examined me said I had heart disease. I thought I would die immediately; he scared me awfully. The next day I went to another doctor and he said he could not find anything at all; that I was perfectly well. I am very nervous, and last Saturday when I felt a pain in my heart I went to a third doctor, who told me I had a little affection of the heart, to go home and stay in bed for a week and see how I would feel. Then I called on Dr. Hamburger. Now, what have I?

DR. HAMBURGER: This man is a tailor and has had an exceedingly hard winter; for months at a time he has not worked. He has always worried a great deal about his family and financial affairs. He wanted to join a lodge and the examining doctor told him he would not be accepted because he had a heart affection. When I saw him Thursday he was in bed, looking about the same as he does now. I will give you the findings:

He had a rapid pulse, 110; this morning it is 120. His heart was enlarged a trifle, out to the nipple line. He had a soft systolic blow at the apex. Nothing in the aortic or pulmonic regions. He suffered no pain; there was no edema; he looked apprehensive, scared. He had seen several men who advised that he could not enter the lodge, and he became very much frightened. Another doctor told him he had nothing wrong with him. The third doctor told him he had not much wrong, but that he better stay in bed. Now he comes to the fourth doctor. Before we discuss this phase any further I want you to listen to the soft blow at the apex. I also want you to decide what opinion you would give him. You may accept from me that his other findings are negative. His blood-pressure is 160. His urine is all right; liver and spleen not palpable; no edema of ankles, and no other complaints or findings.

VISITOR: I would keep him in bed for a couple of weeks; I don't think there is much wrong.

ANOTHER VISITOR: How often do you have pain around your heart? You said Saturday you had pain. Is that the first time?

PATIENT: Last week, Wednesday, I had pain.

DR. HAMBURGER: Never had it before?

PATIENT: No.

VISITOR: Under what conditions does this pain come; were you walking or running?

PATIENT: I was just walking pretty fast.

DR. HAMBURGER: What were you doing when the pain came?

PATIENT: I was working all the time, nothing particular.

VISITOR: I should see if he would improve with rest. I would keep him in bed and watch him for a while.

DR. HAMBURGER: How definite a statement could you make to him now?

VISITOR: It sounds like a mitral regurgitation, but it is not exactly that.

DR. HAMBURGER: What is the matter with him?

ANOTHER VISITOR: I think it is purely physiologic; it is only a functional murmur.

DR. HAMBURGER: What would you do with him?

VISITOR: I would have him rest for a while.

ANOTHER VISITOR: I think it is more of a nervous manifestation.

DR. HAMBURGER: You don't think he has an organic trouble? What would you do with him?

ANOTHER VISITOR: I would get him a job and stop his worry.

DR. HAMBURGER: He has a job now. With the coming of spring he got work.

VISITOR: Does he smoke?

DR. HAMBURGER: He smokes somewhat immoderately; eight to ten cigarettes a day.

ANOTHER VISITOR: I think he has arteriosclerosis.

VISITOR: I think he should not worry. He looks fine.

DR. HAMBURGER: Would you keep him in bed?

VISITOR: I would let him work.

ANOTHER VISITOR: I think there is a mitral insufficiency.

DR. HAMBURGER: Would you let him go to work?

VISITOR: I don't think his form of occupation would hurt him if he stopped worrying.

DR. HAMBURGER: What is the matter with him? What would you advise? Has he a mitral insufficiency? Should he go to work?

VISITOR: It would be all right for him to work, but he should rest as much as possible. He should not overexert himself.

DR. HAMBURGER: Why?

VISITOR: To keep all the strain off the heart on account of the valvular disease.

DR. HAMBURGER: What is the matter with him?

VISITOR: There is no accentuation of the second pulmonic; there is no murmur; the whole history makes one think of a functional condition. I would advise him to work and tell him to take it easy. If he worries, it might make matters worse.

ANOTHER VISITOR: I think it is a functional murmur and I think he can work all right. I would advise him to give up cigarettes, but I would let him work and not worry.

DR. HAMBURGER: What is the matter with him?

ANOTHER VISITOR: I think it is a functional murmur; I think he ought to be watched for further developments.

DR. HAMBURGER: You would let him work?

VISITOR: Yes. But to take it easy at the same time.

DR. HAMBURGER: All right, Mr. C. You may come back here next Saturday and let us see you again. (Patient excused.) He is not as simple a case as may first appear. I will tell you briefly what I found when I first saw him:

He has surely an organic mitral insufficiency. I don't think there is any doubt about that. The fact that this murmur occurs at the apex and not at the base speaks against functional murmurs. His high blood-pressure may make you think that in all probability he has some slight valvular defect. Now, the question is, Is the heart muscle affected? Has he any sign of cardiac insufficiency? We have said many times that a patient can have a valvular lesion with no discomfort. This man is a type that is profoundly neurotic. Anything you tell him will impress him. You must tell him flatly that it is nothing serious. Beginning heart muscle insufficiency is the whole question in this case. When I saw him first I felt convinced the whole thing was a neurosis. He never had shortness of breath; he had no pain. Before the lodge doctor saw him he had no palpitation, no choking sensations, absolutely no sign of cardiac insufficiency. He could run for a street-car or walk up flights of stairs with no discomfort; no swelling of ankles. I attributed his rapid pulse to his neurosis, in spite of the fact that his heart is a bit large, and I gave him bromids; and he comes back better, but he is still profoundly nervous and his pulse is even faster. He has had no more pain. It is a difficult problem to say today that he has or has not some slight involvement of the heart muscle. My personal belief, from experience in similar cases, is that he has not. I still feel certain that he has simply a neurosis. But we should watch him for a week or two, giving him plenty of fresh air and proper food. First of all, his race: he is a Russian Jew, and the Russian Jews are of a neurotic temperament; then his misfortunes, due to hard times, sickness in his family, etc., are contributing factors. I also forgot to mention that he worked thirteen and fourteen hours a day (fourteen hours at tailoring), which, of course, means sitting on a bench with no

exercise. These I believe to be the true factors of his neurosis.

However, it is well known that overtire and overwork will produce slight heart muscle insufficiency, and it is possible that he may have that. If he has, then rest and quiet and freedom from worry and strain, cutting down work, abandoning cigarettes, toning up the heart muscle, etc., are indicated. I do not think it makes much difference whether his condition is purely a neurosis of the vagus or sympathetic nerves or whether he has a very slight heart muscle involvement, because, to all practical purposes, the treatment is the same. If he is kept in bed he worries and is very much worse. It does not matter much whether this is purely a neurosis or the result of poor hygiene, worry, and the lack of exercise. The fact remains that he has a tachycardia, and that he has possibly a little heart muscle involvement. The important thing for us to do is to observe him properly and to advise him properly.

As several men suggested, theoretically, this man ought to be kept in bed; his blood-pressure would go down; but he is a man who begins to worry when you keep him in bed and you must individualize your cases. His wife told me that if he worked he would worry less and then he seems to be in better shape. Problems just like this—the early stages of cardiac disease—are the cases you meet with in practice, private or hospital. If this man goes on working improperly, living improperly, his heart muscle might begin to give out. I believe it is important in training medical men to teach them not to frighten people. Just because he has an organic valvular leak his whole life should not be spoiled by fear. If you had been examined, say five or ten years ago, and some doctor had found a heart leak and had told you not to overstrain and to be careful of your activities, it would have had a profound influence upon your life. It is important, particularly in heart cases, to guard your opinion. If the individual is actually seriously ill it is probably better to tell the other members of the family, because the psychic and nervous effect, especially in neurotics, is very profound. We will

try to have him come back from time to time and see how he is getting along.

VISITOR: Does any exercise influence the murmur?

DR. HAMBURGER: There was no change in the murmur. That could be tried, however.

VISITOR: You would not give anything for the heart at all; bromids, just to quiet him?

DR. HAMBURGER: Yes. On examining him I thought of several things. In addition to the murmur he has a tachycardia, and one remembers that tailors get certain diseases, such as tuberculosis, etc. His high blood-pressure speaks somewhat against tuberculosis. In going over his lungs he has absolutely nothing. No temperature; no night-sweats; no cough; nothing to suggest tuberculosis. Likewise, in Basedow's disease you get a rapid pulse; he has a slight tremor, but he has no other signs of exophthalmic goiter. In very early heart insufficiency, you remember, we spoke of the tests of Moritz, that when a given tachycardia disappears in sleep it speaks for a neurosis; when it persists, for an organic muscle lesion. That could be tried out if we had him in the hospital.

There are certain things in his condition that make one suspect beginning organic muscle change—the high blood-pressure, the tachycardia, etc. As I have said, the ideal thing to do would be to keep him in bed.

NOTE.—The patient did not return to the clinic and was not observed again. However, a communication from his wife, six weeks later, stated that the patient had entirely recovered from his cardiac complaints, had gained in weight, and was working with his usual strength.

AURICULAR FIBRILLATION; A CLINICAL ENTITY

THE patient we wish to present this morning has been chosen for the purpose of demonstrating the typical findings of a condition which has only been recently described as a distinct clinical

entity, namely, auricular fibrillation. The patient's history is as follows:

This man has been a steeplejack for years. He comes to the hospital with the following complaints: Pain in his left side, chest, and back; onset six weeks ago with a heavy cough; he took a cough syrup which the doctor said contained strychnin and nitroglycerin. Since then, particularly the last two weeks, he has suffered from increased dyspnea on the slightest exertion and pain in his side and back intermittently. *Past history:* He had a heavy cold four or five years ago and was told he had overtaxed his heart. He became better, and then became dyspneic, until his recent attack. He has never been ill otherwise. He has been a fairly heavy drinker; has used nicotin to excess, practically smoking or chewing all day long. He had gonorrhea when a young man and chancre when thirty years of age. He is fifty-five years old. Until five years ago he had no subjective cardiac complaints; then he caught a severe cold and his doctor told him he had a heart involvement. Two years ago he began to be dyspneic, and now he has marked dyspnea upon the slightest exertion, and he complains of pain in the chest and back.

DR. HAMBURGER (addressing Patient): What kind of pain is it?

PATIENT: Heavy; it is like a ball of lead in there (pointing to heart). When I rub it it seems as if I was making more room for my heart.

DR. HAMBURGER: Does the pain go to your shoulder?

PATIENT: It goes over to this side (pointing to left side).

DR. HAMBURGER: Does it come on more at night or during the daytime?

PATIENT: It comes on in spells.

DR. HAMBURGER: On examination, we see a man past middle age, who was somewhat cyanotic and dyspneic six weeks ago on admission, but who is not especially dyspneic now; his condition in this respect is quite different from when he entered. The scalp is negative except for scars received when injured in his work as a steeple climber; his eyes are normal; teeth negative; throat negative; respiration slow and shallow.

PATIENT: When I sit up I feel all out of breath.

DR. HAMBURGER: He has marked shortness of breath. The apex-beat is visible now in the sitting posture. At the time of admission, six weeks ago, the right border of his heart was outside of the midsternal line, left border outside the nipple line. There was great tenderness over the spleen and some over the liver, both slightly palpable; no tenderness elsewhere. An old scar (former chancre) is present. The diagnosis when he came in was myocarditis. He was put on tincture of digitalis on the day of admission and has been on it ever since.

PATIENT: I am getting 5-drop doses of digitalis. I have been taking it two years.

DR. HAMBURGER: Now, on observing him, you see marked dyspnea on exertion. You will particularly note his pulse, which is rather slow, but markedly irregular. The intervals between beats are irregular, sometimes extremely long and sometimes very short; in addition, there is marked irregularity in the strength of the individual beats. The pulse, graphically, would look something like (Fig. 67). For a while regular and then several beats coming together, then a large one, then another large one, and then a complete irregularity; both rate and strength are irregular (Fig. 68). Such a completely disordered pulse is characteristic of auricular fibrillation, the pulsus irregularis perpetuus.

On continuing the examination, you will note the finger-nails have pretty good color; his pupils are equal; both eyes show a pronounced arcus senilis. Lungs, posteriorly, are clear; no râles at the base. Anteriorly, perfectly clear, no râles. His heart extends 3 cm. to the right and 13 cm. to the left. The upper border is at the third rib. He has no substernal dulness, no areas of pulsation, no tracheal tug. There is no palpable thrill; no murmurs to be heard. The second aortic is slightly accentuated. The irregularity is the most important phenomenon noticed on auscultation. There is slight tenderness over both liver and spleen. No edema of ankles.

Then to summarize: He has no findings, on physical examination, except the marked dyspnea on slight exertion, slight cyanosis, increase of heart to the left and slightly to the right,

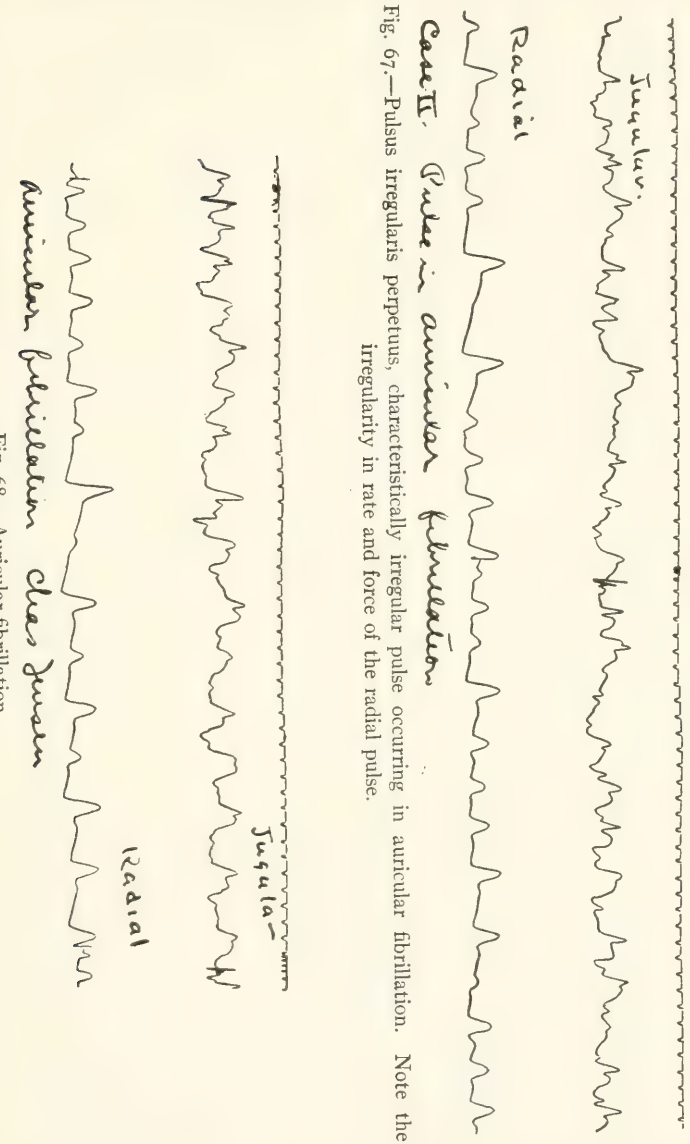

Fig. 67.—Pulsus irregularis perpetuus, characteristically irregular pulse occurring in auricular fibrillation. Note the irregularity in rate and force of the radial pulse.

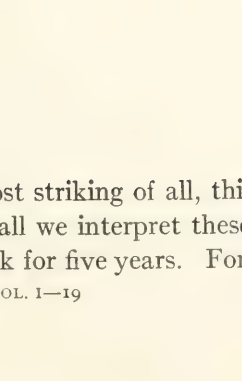

Fig. 68.—Auricular fibrillation.

and, most striking of all, this complete irregularity of the pulse. How shall we interpret these findings? Here is a man who has been sick for five years. For two years he has had marked dysp-

nea; he has been taking digitalis for two years; no murmurs. What is the matter with him?

VISITOR: There probably was a murmur there first.

DR. HAMBURGER: Why?

ANOTHER VISITOR: Has he myocarditis?

DR. HAMBURGER: If it is myocarditis, what kind is it, acute or chronic?

VISITOR: Two years would be chronic.

DR. HAMBURGER: He has a myocarditis, there is no question about that; chronic myocarditis with fibrous changes in the heart muscle, with associated arteriosclerosis.

VISITOR: How about the irregularity?

DR. HAMBURGER: That is really the only striking sign he has. He has dyspnea and pain. He has no loud, coarse murmur; he has a markedly irregular pulse, and it is rather slow, although on entrance it was very fast, but he is now on digitalis.

Auricular fibrillation has only been described within the last five or six years, and is now almost universally accepted. There is no doubt that it will be classified as a clinical entity. It occurs in myocarditis, but also without. The first work on auricular fibrillation was done by Mackenzie in 1890, when he studied the irregularity of the pulse.

Mackenzie, in studying cardiac irregularities, tried to differentiate them into groups, and conceived the idea that tracings from the jugular vein might give some light on the significance of these cardiac irregularities. Therefore, before we can talk on this subject we must have some idea of the normal jugular pulse as obtained by the polygraph. There are usually three waves which are important to define. The tracings look something like this (Figs. 69, 70). These waves have been named *a* wave, *c* wave, and *v* wave. The *a* wave has been shown to be due to contraction of the auricle. As to the causation of the *c* wave there is some difference of opinion. Mackenzie believes it is possibly due to the closure of the semilunar valve, but whatever the actual causation, it occurs during the contraction of the ventricle. The *a* wave is due to the contraction of the auricle; *c* wave to the contraction of the ventricle; the interval between

them, that is, the time necessary for conduction of the wave of auricular contraction to the ventricle, amounts to $\frac{1}{5}$ second; that

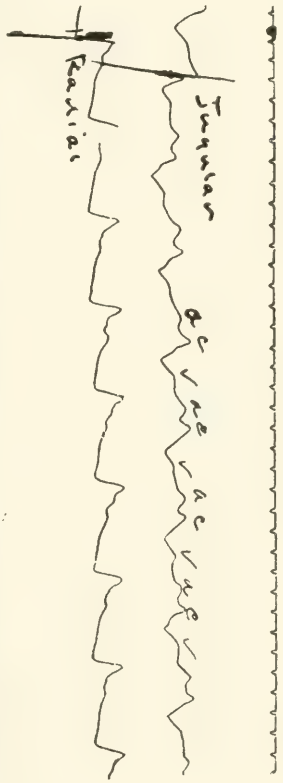

Fig. 69.—Normal radial and jugular pulse tracing. Upper line, time marker beating fifths of a second. Note appearance and relations of *a*, *c*, and *v* waves in jugular tracing.

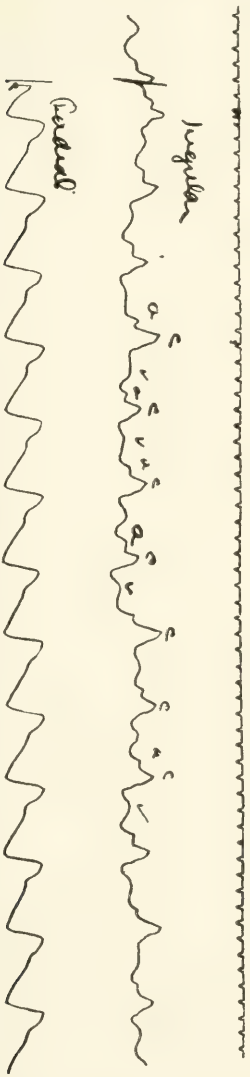

Fig. 70.—Normal pulse tracing, showing characteristic waves.

is, the *a* to *c* interval is $\frac{1}{5}$ second. We will go into this more fully another time. The *a* wave is auricular contraction; *c* wave is

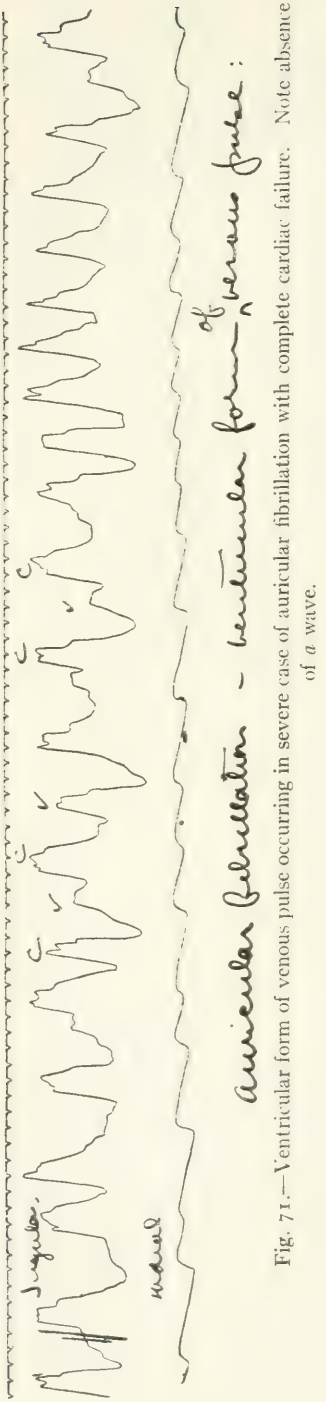

Fig. 71.—Ventricular form of venous pulse occurring in severe case of auricular fibrillation with complete cardiac failure. Note absence of *a* wave.

ventricular contraction; *v* wave is a little more difficult to define in simple terms, but it is thought to be due to closure of the tricuspid valves with temporary stasis of blood in the right auricle and jugular just before the blood in the right ventricle is emptied. So the *v* wave is due to closure of the tricuspid valve plus stasis.

Mackenzie found in about 60 to 70 per cent. of cases of irregularity that the *a* wave was absent, so that the tracings would simply show the *c* and *v* waves. Such a venous tracing is spoken of as the ventricular form of venous pulse (Fig. 71).

Now what is the explanation of the absence of the *a* wave? Mackenzie at first thought the auricle and ventricle contracted simultaneously, and as a result of that conception he spoke of this irregularity as *nodal rhythm*. The idea was that the auricular contraction really disappeared and only the ventricular contraction took place.

The subsequent work of Lewis showed that this conception was, in all probability, erroneous. He demonstrated that this irregularity can be produced by stimulating the auricle experimentally and causing it to fibrillate, thus

producing auricular fibrillation and disappearance of the *a* wave.

What do we mean, clinically, by auricular fibrillation? It occurs in two classes of individuals: *first*, in mitral stenosis. A young individual who has had an acute rheumatic infection subsequently develops an acute endocarditis of the mitral valve, with secondary mitral insufficiency and stenosis. Following the regression of the acute infectious process the mechanical stenosis remains, and later the heart muscle becomes involved, with the early signs of incipient heart muscle insufficiency. In such an individual, with persistence of the mitral stenosis and development of heart muscle insufficiency, auricular fibrillation frequently sets in. Why? In all probability the mechanical narrowing of the mitral orifice throws an extra burden on the muscle wall of the auricle, already diseased, with the result that fibrillation of the auricle occurs.

Second, the senile heart, that is, fibrous myocarditis. For example, an elderly patient with chronic fibrous changes in kidney, blood-vessels, heart muscle, and secondary degenerative changes associated with hypertrophy and dilatation of the heart, develops signs of heart muscle failure. Early in its development an irregularity of the pulse appears, usually of the type of auricular fibrillation.

In all probability when this particular patient came in he had some involvement of his myocardium. Probably all cases that have auricular fibrillation have some heart muscle change, and that is why it was important in the previous case of cardiac neurosis to know whether or not he had an early heart muscle change, because at some time he may develop auricular fibrillation; but the heart muscle in mitral stenosis following rheumatism is a little bit different.

Now a word about the occurrence of auricular fibrillation and the significance of it:

Men who have observed it long, Mackenzie and Lewis particularly, agree that it occurs under many conditions. It occurs as a transient phenomenon in many infections—diphtheria, pneumonia, etc. It has been brought out by digitalis where before it

was latent. In pneumonia, diphtheria and typhoid, or any acute infection, auricular fibrillation is likely to be transitory. For example, in the case that Dr. Foley showed you of auricular fibrillation in pneumonia, that probably occurred acutely with the pneumonia, although it may have been present before and been constant. If it disappeared after the pneumonia we know that it was an acute attack. Auricular fibrillation may occur abruptly once and become permanent. More frequently, however, it occurs and disappears, then recurs and recurs, and finally becomes permanent.

Auricular fibrillation may cause symptoms similar to other cardiac affections or it may be latent. Patients very frequently complain of a rapid pulse; it may be slow, but usually is rapid. They complain of palpitation. Then the heart is enlarged on account of the hypertrophy and consequent dilatation. The irregularity may persist for a long time with no other special signs of heart disease. The heart may be functionally competent, but more classically the patients have marked broken compensation and usually edema. This man had most of these symptoms when he came in, and is now improved by digitalis.

This case, then, will serve as a fairly typical example of auricular fibrillation. Its interest and importance may easily be judged from the fact that 60 to 70 per cent. of all cases of serious heart failure met with in practice are cases of auricular fibrillation. The pulse of auricular fibrillation may be defined as an irregular pulse of a very disordered kind, and is that form of irregularity so frequently met with in the elderly, and in patients with hearts damaged by previous rheumatic affections, particularly in mitral stenosis. In fact, some writers call it the "mitral pulse." There are various terms used synonymously to describe this pulse which you ought to know: "Completely disordered pulse" and the "Pulsus irregularis perpetuus," the "P-I-P" pulse, etc. In the long-standing classical cases of serious heart failure, auricular fibrillation is usually accompanied with dropsy and extreme dyspnea.

What is auricular fibrillation? The answer to this question may be concisely stated in the terms of Mackenzie:

"The term 'fibrillation' is applied to a curious condition of the muscle-fibers of the heart where the individual fibers in place of contracting in an orderly and simultaneous manner during systole contract rapidly and independently of one another. The auricle when in a state of fibrillation presents an entirely different aspect from what it does during its normal action (Lewis). The walls of the auricle stand in the diastolic position. Systole, either complete or partial, is never accomplished. The wall, as a whole, is stationary, but careful examination of the muscle reveals an extremely active condition; it appears to be alive with movement; rapid, minute, and constant twitchings or undulatory movements are observed in a multitude of small areas upon its surface."

Prognosis of Auricular Fibrillation.—Let us speak for just a moment of the prognosis in auricular fibrillation. In the first place, the time of occurrence of the fibrillation is of some importance. For instance, an acute attack of auricular fibrillation may occur and subsequently disappear, never to come back, or it may be simply the first attack with many subsequent recurrences. I should say the rule is that it tends to recur many times, finally, in long, severe cases becoming permanent. The prognosis, as far as expectancy of life is concerned, must be based on two points. *First, the response to treatment:* It is well enough before venturing an opinion in auricular fibrillation to see how much can be done by treatment. In other words, how much unimpaired heart muscle is left possessing the ability to respond to proper medicinal and hygienic treatment. Good results may be obtained by the use of digitalis and rest, where, before the effect of such treatment had been observed, the outlook may have been gloomy indeed. In other words, no safe opinion can be given until the patient has had the benefit of proper and prolonged treatment.

Second, the response to effort, that is, the testing out by proper methods, of heart muscle *sufficiency* before treatment has been instituted. The fact that any given patient may show the findings which we have now learned to recognize as auricular fibrillation is no absolute indication of the condition of his heart muscle; although he may have completely disordered rhythm, he may be

able to perform the work and duties of his life without dyspnea, without cyanosis, without swelling of the feet and ankles; in short, without any definite evidence of beginning heart muscle failure. In such an event the prognosis would be favorable, showing that he has enough heart muscle remaining to give him sufficient circulation for performing the ordinary duties of life. In other words, if response to effort and ability to work are good, the prognosis should be good. On the other hand, if he cannot perform the ordinary tasks of life without signs and symptoms of cardiac impairment, the prognosis is relatively bad.

Treatment.—The treatment of auricular fibrillation marks one of the real epoch-making advances in the modern treatment of cardiac disorders. If the intensive study by Mackenzie, Lewis, and others had resulted in no other findings than the positive indications for digitalis in auricular fibrillation, it would have been a most valuable discovery. It may safely be said that the most brilliant results of cardiac therapy may be obtained by the proper administration of digitalis in cases of auricular fibrillation. Again, to quote from Mackenzie:

"It is seldom that I have been able to say that I have saved a patient from immediate peril by the use of drugs, but this I can say with confidence, that I have repeatedly seen patients in evident peril of death removed rapidly from danger and restored to a condition of comparative health and fit for work by the judicious use of digitalis."

The success or non-success of treatment may be said to rest almost entirely in the knowledge of dosage of digitalis and the proper adjustment of the drug to the individual case. This point should be emphasized as of greatest importance, that whichever of the many preparations of digitalis is used, *the drug should be given to a point of sufficiency*, that is, enough digitalis should be given until you are positive that you have obtained a physiologic effect. Under digitalis, given in proper dosage, the rapid and completely irregular pulse of auricular fibrillation very promptly falls in rate and becomes regular.

My personal preference is the tincture, given in from 10- to 20- or even 30-minim doses four times daily, starting with large doses at the beginning of the treatment and then only diminishing the dose as sufficiency is approached. An adult patient presenting himself with the classical signs of broken compensation, namely, a completely irregular pulse, orthopnea, enlarged and tender liver, ascites, edema of the extremities, cyanosis, a pulse ranging from 110 to 140 or more, may be given 1 or $1\frac{1}{2}$ drams of the tincture daily. After from four to seven days of such dosage, *provided the digitalis is absorbed from the congested mucous membrane of the gastro-intestinal tract, the pulse should drop from 140 to 120, 100, 90, 80, 70.*

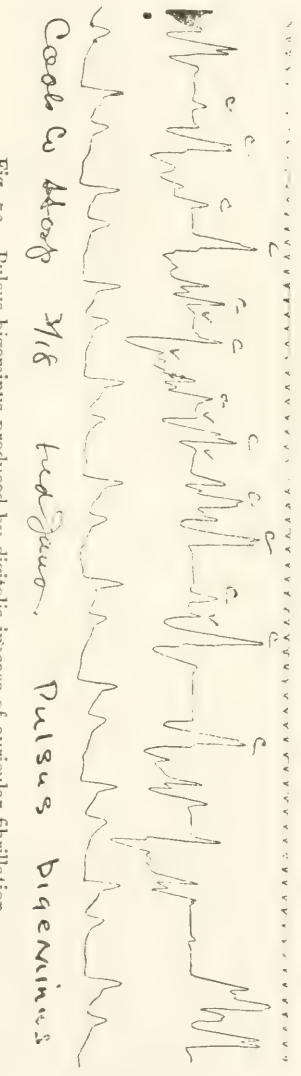

Fig. 72.—Pulsus bigeminus produced by digitalis in case of auricular fibrillation.

Let me emphasize here that we are speaking of the ventricular pulse-rate and not of the radial, for it is well known that in a completely disordered pulse, with varying strength of ventricular contractions, the ventricular rate may be 140 or more, the radial 90 or less. As the pulse-rate is slowed, digitalis may be gradually diminished or stopped. If you have discontinued digitalis with the pulse about 70, careful watching of the pulse is necessary, for in the event that it starts to rise, reaching 80 or 90, digitalis should again be started, in somewhat smaller doses. Continue the administration of the smaller dose until you have ascertained as nearly as possi-

ble the exact amount of digitalis required by this individual patient to keep his pulse constantly at its most efficient rate, in most instances 70 or 72. Pardee,[1] in a recent article, emphasizes this particular point.

One word of warning is necessary when giving large doses at the start: Careful daily observation is necessary to detect the first signs of toxicity. These signs may be stated to be the subjective complaints of nausea and vomiting or other gastro-intestinal distress, and headache or ringing in the ears; objectively, the production of a bradycardia (pulse under 60) or of a bigeminal pulse (Fig. 72). The margin of safety between the physiologic and toxic limits is sometimes very small, so that extreme care and observation are necessary. Failure to exercise such care may result disastrously, even in death. If the particular preparation given by mouth is not being absorbed it may be necessary to resort to intravenous medication, using digipuratum or digalen or strophanthin.

Of course, there is a great deal more to be said: for instance, we used to think that digitalis raised the blood-pressure, and therefore it was dangerous to administer in cases of high tension. This probably is not true. In the presence of embarrassed circulation, with heart muscle insufficiency, digitalis should be given, irrespective of the findings of the sphygmomanometer.

IRREGULARITIES OF THE PULSE, IMPORTANCE OF THEIR RECOGNITION AND DIFFERENTIAL DIAGNOSIS, PARTICULARLY FROM THE STANDPOINT OF TREATMENT

Dr. Hamburger: I want you to see this boy who entered the dispensary complaining of constipation. Use your stethoscopes and have him take a slow, deep breath. I brought over

[1] The Prognosis of Auricular Fibrillation, Harold E. B. Pardee, Jour. Amer. Med. Assoc., 1915, lxiv, 2057.

several tracings of these various irregularities that I want you to see.

Sinus arhythmia is an irregularity of the pulse which occurs most frequently in young individuals, particularly at puberty. It is not due to diseased heart muscle, but is due to variations in the vagus control of the heart. The vagus nerve comes down and centers about the sino-auricular node at the junction of the superior and inferior venæ cavæ. The node is often spoken of as the "pacemaker of the heart"; first described by Keith and Flack. This "pacemaker" is directly under the control of the vagus. Stimulation of the vagus produces bradycardia. Why? Because these fibers are inhibitory, thereby giving a slower pace to the heart than normally. So that as the inhibitory action is increased you may get a bradycardia, and as the vagus influences are decreased you get a normal pulse or even a tachycardia. Now in certain individuals, particularly in children up to the age of puberty, a certain type of irregularity, called *sinus arhythmia*, occurs, which is not in any way related to the heart muscle. The most outspoken cases show respiratory arhythmia. At the time of deep inspiration with the descent of the diaphragm the heart increases in rate because of the lessening of the vagus control; with expiration, the normal rate returns. Experimentally placing one's hand on a dog's chest at the time of inspiration, you feel the heart as it goes faster, and when the vagus gets control again you get bradycardia. Sinus arhythmia occurs physiologically in children; it is usually not a true arhythmia, and may occur in certain older individuals, especially in neurasthenics.

This boy is seventeen years of age, and demonstrates an example of outspoken sinus arhythmia. Its importance is mostly its recognition. It is simply an expression of vagus control. Leave it alone. Don't treat it. Giving digitalis to a sinus arhythmia is, of course, worse than folly. It is like giving a lot of medicines and drugs to an entirely normal individual. Now this boy is a classical type of sinus arhythmia at puberty. He came to the dispensary complaining of pain, with a history of constipation. Notice when he breathes his pulse gets a bit

weaker, and at the height of inspiration, there is no pulse at all. Did you get that? His pulse disappears and disappears as long as he holds his breath. Now what happened during the disappearance of the radial pulse?

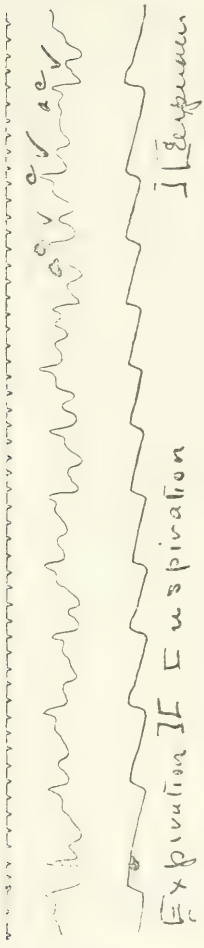

Fig. 73.—Sinus arhythmia in a man thirty-eight years of age. Note increased rate on inspiration, decreased on expiration.

VISITOR: Either the heart stopped beating or the heart continued beating and the pulse could not be felt.

DR. HAMBURGER: Although the radial pulse disappears, you will note upon listening at the apex that the contractions of the ventricles go on quietly and regularly. It is not a complete standstill of the heart; and, moreover, he is perfectly comfortable; so the remote possibility that the heart is stopped is, of course, ruled out. The disappearance of the pulse has been called "pulsus paradoxicus." This boy has not been ill; he has no temperature; he is at the age of puberty and has a disappearance of the pulse upon inspiration. The "pulsus paradoxicus" also occurs in neurasthenics. Now to explain why it disappears: It is caused either by the contraction of the diaphragm, pulling on the pericardium, thereby interfering with the arterial pulse, or is due to the dropping down of the diaphragm on inspiration. There may be other explanations. At all events, it is certain that the heart goes on beating perfectly regularly.

I have some tracings here which I will pass around showing a case similar to this. This shows jugular pulse above and radial pulse below (Fig. 73).

Now simply to emphasize the importance of sinus arhythmia: Get a clear conception that it is an affair of the vagus and not of the heart muscle, and that it should be left alone. Don't treat such a case with digitalis; send him about his business; give him hot and cold showers to improve the tone of the nervous system, but *don't treat him with digitalis.* I should say that 90 per cent. of all the irregularities occurring in children should not be treated; also 90 per cent. at the age of puberty should not be treated. This boy complains of cardiac pain, but there is no cardiac insufficiency. Simple out-door exercise with hot and cold showers—anything to improve the tone of the nervous system. It will probably persist with him more or less during his life, but he should not be treated for "heart disease."

The question of cardiac irregularities we took up last Saturday for the purpose of demonstrating one of the commonest types, an auricular fibrillation. We said that auricular fibrillation was one of the commonest types of irregularity in grave heart lesions, occurring in about 60 per cent. of heart failure. Now, although that is one of the common types of irregularity, there are many others, and we are to be given a little summary of the most common types of cardiac irregularities, and after that we will discuss the further points that we omitted, and thereafter discuss this patient here, who has a distinct and suggestive type of irregularity.

The pulse is an expansion and relaxation of the arterial wall produced by the intra-arterial pressure changes at that point. There are various changes in the normal rate, as in man the average pulse-rate is 70; in woman, 80; and at birth and all through the different stages of development, from childhood to old age, there is a change in the pulse-rate. At birth the pulse-rate is usually 140; in infancy, 120; in childhood, 100; in youth, 90; in the adult, 75; in old age, 70; and in extreme old age it gradually rises again, 75 and 80. Then the variations, both the increase and decrease: the increase appears normally after meals, with low blood-pressure, muscular exercise, standing up, and in cases of asphyxia where carbon dioxid is increased, and this is followed shortly after by a decrease in the pulse.

In the form of normal pulse I have shown here on the board, "p" is the predicrotic wave and "d" the dicrotic wave—those are the pulse waves. The dicrotic occurs when the blood-vessels are dilated and during ventricular contraction. Under abnormal con-

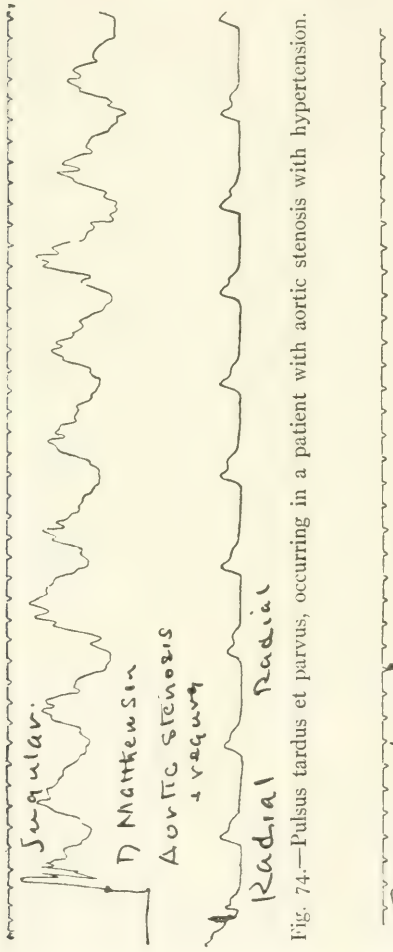

Fig. 74.—Pulsus tardus et parvus, occurring in a patient with aortic stenosis with hypertension.

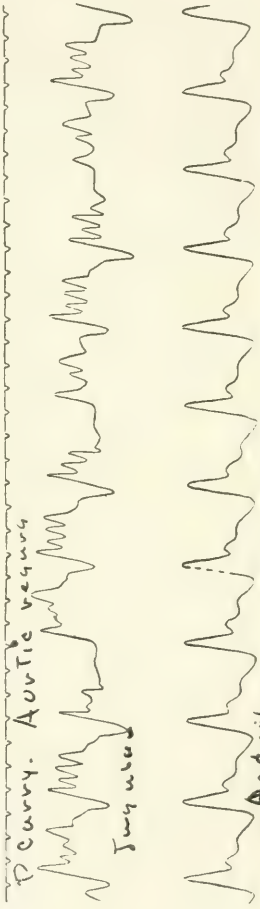

Fig. 75.—Pulsus celer et alter, or Corrigan pulse, occurring in aortic regurgitation.

ditions the dicrotic pulse may be emphasized and palpated with the finger. The dicrotic pulse results from closure of the semilunar valves. The anacrotic limb does not show an extra wave except in arteriosclerosis and aortic stenosis. The pulse may be irregu-

lar, both in force and rhythm. The former, that is irregularity in force, is a serious condition, if overexertion and temporary toxic influences can be ruled out. It is usually more serious in connection with a lesion of the aortic valve than in mitral disease. It often occurs in connection with sclerosis of the coronary arteries and myocarditis. Then there is one type of pulse, high-tension pulse, that is very difficult to compress. This is what it is (illustrating on board; see Fig. 74). It has a wave of moderate height; falls away slowly. It is caused by cold; old age, pre-eclamptic and eclamptic stage; hysteria and migraine; cerebral hemorrhage; chronic nephritis and arteriosclerosis, and in aortic stenosis.

Then, in contradistinction to that, there is a low-tension pulse, where you have a sharp summit and the pulse collapses between the beats, and this is characteristic of the "Corrigan pulse." This is caused by a weak heart with low arterial tension or else in cases of aortic regurgitation (Fig. 75). The "bounding pulse" of an early infectious process has this appearance (illustrating low-tension pulse on board). In mitral regurgitation the pulse is normal until late in the disease; then it becomes irregular in force and rhythm. In mitral stenosis the pulse is small and out of proportion to the apex-beat. Late in the disease it becomes irregular. The pulse is very similar to a pulse of high tension, but bigger (illustrating on board). In aortic regurgitation we have this type (illustrating on board), low tension, that is, the Corrigan pulse, and another term for this low-tension pulse is the "water-hammer" pulse and "pulsus celer." Pulsation is marked all over the body. The pressure rises sharply under the examiner's finger and falls away as abruptly. This is due to a large volume of blood being forcibly and suddenly thrown out by the hypertrophied and dilated left ventricle. As the artery empties itself in two directions the marked fall is evident. Then in aortic stenosis you have this type (illustrating on board), long and sustained, and known as the *pulsus rarus*, *tardus*, and *parvus* (see Fig. 73). The pulse rises very slowly. There is no big pressure to force it up high. This pulse is only recognized in uncomplicated aortic stenosis and is obliterated if aortic regurgitation is also

present. It sometimes occurs in mitral stenosis and renal disease. In aneurysm one difference is that the left pulse is delayed after the right.

Dr Thomas Lewis records other irregularities of the pulse. The first type is the solitary pulsus intermittans, and the cause of this is premature contraction; the second, dropped beat or heart-block; next, coupled pulse beats, "pulsus bigeminus," or tripled beats, "pulsus trigeminus." This pairing of pulsations may result from premature heart-beats. The next is the grossly irregular pulse, where you have a jumbling of pulsations and imperceptible beats, and this is usually due to auricular fibrillation. Then the alternation of the pulse, "pulsus alternans," is seen and shown by this diagram, the upper line (indicating on diagram) representing the auricular beat and the lower the ventricular beat. This is seen in two classes of patients: Those whose heart rate is unduly accelerated, as in paroxysmal tachycardia, and, second, it occurs when the heart rate is within normal limits. This is observed in elderly people, chiefly males. Other causes are angina pectoris, high blood-pressure, renal disease, fibromyocarditis. "Pulsus alternans" is thought to be due to healthy heart muscle carrying an excessive load or degenerated muscle struggling to perform work of which it is barely capable. In most cases this pulse can be duly recognized with instruments.[1] It is not to be confused with the dicrotic pulse, for in the latter the pulse-beat is twice the rate of the ventricle. The subjective symptoms with this type of pulse are anginal pains, breathlessness (usually nocturnal), periodic dyspnea.

Prognosis for the patient with "pulsus alternans" is bad; unexpected death is the common termination. To quote Dr. Lewis: "It is the faint cry of an anguished and fast-failing muscle, which, when it comes, all should strain to hear, for it is not long repeated—a few months, a few years at most, and the end comes."

[1] Several writers have recently called attention to the use of the blood-pressure cuff (sphygmomanometer) for the identification of pulsus alternans—alternating beats coming through the compressed brachial artery at different tension levels.

The problem of irregularities of the pulse is probably of greater importance practically than the detection of a valvular lesion, and while the subject is really in its infancy, still a sufficient amount of information has been obtained to make it valuable, and you should have some clear-cut notion of it.

Heart-block.—The "pacemaker," or node of Keith and Flack, at the junction of the vena cavæ discharges nerve impulses over the entire auricle, which are transmitted in turn by the bundle of His to the ventricle; and, as we said in the interpretation of the jugular tracings, the time is about $\frac{1}{5}$ second. So the *a* to *c* wave is $\frac{1}{5}$ second.

Normally, the contraction wave travels from this path through the bundle of His. Here you see the contraction of the auricle (tracing). Now, in certain pathologic conditions there may be an interruption of these conduction impulses through the bundle of His, and instead of each contraction reaching the ventricle, you may have an interference, so that every second or every third impulse is interfered with, so that one impulse may come through in good shape, likewise the second, while in the third you have a drop of the beat or an interference with the conduction of the third impulse. That condition is called a "drop-beat" or heart-block, which may be either partial or complete. In the complete block you have a complete absence of conduction—a complete block of the auricular impulses, the ventricle, setting up its own rhythm—where every beat is blocked from getting through.

ANOTHER VISITOR: How long can that run?

DR. HAMBURGER: For years. The ventricles take on a rate of their own. Of course, as a rule, it does not go on for years. The disease known as Adams-Stokes is a condition of complete block, where, in addition to irregularity of the ventricles, you get a recurring transient anemia of the brain with syncope or fainting and unconsciousness. We will speak of this at greater length later on.

In point of frequency heart-block is very rare. I would say at the most 10 per cent. Premature contractions are much more frequent, occurring in 20 to 30 per cent.

Now what do we mean by premature contraction? Another name is **extrasystole.** It is a contraction of the ventricle or auricle appearing at a premature period in the cardiac cycle. The pulse tracing would show, instead of a perfectly regular recurrent pulse, two beats occurring together and followed by a compensatory pause; the premature beat is followed by a compensatory pause (Fig. 76).

Now, as I said, these occur in 20 to 30 per cent. of cases of irregular pulse. When you feel the pulse of a man over fifty and get a perfectly normal rhythm for one to five minutes and then a little short irregularity—an unusual or extra beat—and then it goes along in regular rhythm again, those are "extrasystoles."

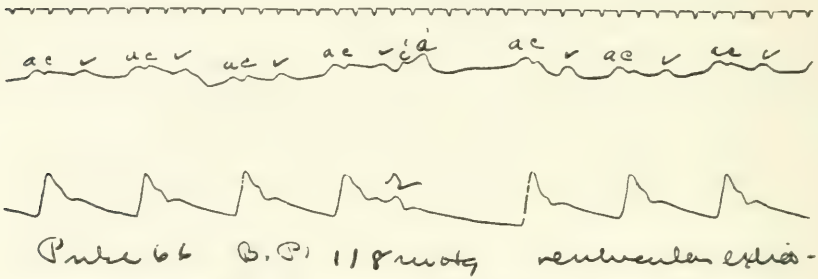

Fig. 76.—Solitary extrasystole or premature contraction-wave *r* occurring in an otherwise regular pulse. Note compensatory pause.

On auscultation in such cases one hears the regular sequence of sounds at the apex interrupted by two short, sharp sounds, followed by a long pause. This finding on auscultation is the easiest and most dependable method of recognition of "extrasystole." Perfectly normal pulse, then a click, just a single escaped beat—those are "extrasystoles."

Premature contractions are usually early signs of myocarditis. The importance is their recognition, to be sure there are no "extrasystoles." They occur in men over fifty.

The treatment is about the same thing as we have discussed in sinus arhythmia. It means a very early change in the heart muscle. Those conditions leading to heart muscle changes should be avoided. Reduce the blood-pressure by rest in bed or drugs

or diet. You frequently get a premature contraction due to nicotin-poisoning in excessive cigar or cigarette smokers. Likewise following periods of overeating or overdrinking, or in individuals who chronically eat or drink to excess, extrasystole irregularities are frequent. The management of such cases revolves about advice and instructions regarding moderation in eating, drinking, and smoking, with general attention and care to personal hygiene. With such supervision extended over a period of months or years (if not permanently) degenerative changes in heart, kidney, and blood-vessels are held in check, and the clinical improvement in the patient is often surprising.

Moderate exercise, such as golf, walking, etc., should be encouraged, as well as frequent vacations and periods of freedom from business stress and worries. The keynote to successful treatment is MODERATION in work, in play, in food and drink, with periods of rest and relaxation alternating with work and strain.

CLINIC OF DR. ROBERT B. PREBLE

St. Luke's Hospital (At Bedside)

A CASE OF MITRAL STENOSIS AND MITRAL INSUFFICIENCY IN A YOUNG GIRL, WITHOUT SUBJECTIVE SYMPTOMS. PREVENTION OF DECOMPENSATION. THE CONSERVATION OF HEALTH AND LIFE THROUGH SCHOOL INSPECTION AND SUPERVISION

This little girl says she is not sick and never has been sick, except that she has had rheumatism several times, but never severely. That is all there is in the history. She is a bright little girl, about twelve years old, is in the seventh grade in school, and was sent in because she attracted the attention of the visiting nurse, who thought she ought to come into the hospital. All my efforts fail to elicit any complaint whatever from her. What do you see there, Doctor?

Intern: The apex-beat is displaced almost to the anterior axillary line.

Dr. Preble: What interspace?

Intern: The sixth interspace.

Dr. Preble: The sixth interspace and anterior axillary line. Is that necessarily pathologic?

Intern: Yes.

Dr. Preble: Yes, there must be something the matter with the child; what?

Intern: Either enlargement of the heart or displacement.

Dr. Preble: The apex-beat is displaced outward as far as the axillary line and in the sixth interspace, and this is probably due to enlargement; can you see anything else?

Intern: The cardiac impulse is diffuse and there is slight bulging of the epigastrium.

DR. PREBLE: I think that is due to the fact that she is not lying straight. Can you see any retraction of the sternum?

INTERN: No, I cannot.

DR. PREBLE: The cardiac impulse is diffuse. How about a capillary pulse? Can you see that?

INTERN: I can't see it very plainly.

DR. PREBLE: It is not very gross, but one can make out a capillary pulse. How about the pulse in the arm?

INTERN: I can't find anything there.

DR. PREBLE: There is a palpable first tone and palpable second tone over the apex. Can you get that?

INTERN: Yes, I do now.

DR. PREBLE: You try it, Doctor. They are rather more palpable than they ordinarily are, so you had all better try it. A very light pressure is best, a mere contact. Have you made a diagnosis?

INTERN: I was thinking of mitral stenosis.

DR. PREBLE: What makes you think of that?

INTERN: You say there is a thrill there.

DR. PREBLE: A systolic; what has that to do with mitral stenosis? When you have a thrill, what part of the cycle is that?

INTERN: The diastolic portion; the thrill might be carried over.

DR. PREBLE: Why not say that it is mitral insufficiency?

INTERN: I did not think there was a thrill in mitral insufficiency.

DR. PREBLE: Do you get a thrill, Doctor?

INTERN: I wouldn't gamble on it.

DR. PREBLE: Use very light pressure; strong pressure will obliterate it. Can you get a systolic thrill at the apex over a normal heart? In other words, is that systolic thrill that we feel necessarily pathologic?

INTERN: I don't know about that, but it is usually pathologic? I don't think you would get a thrill unless there was something pathologic.

DR. PREBLE: That is the common idea, but it is not a true one; you can get it over a perfectly normal heart. I am talking

about the apex now; at the base it would be more likely to be pathologic.

INTERN: Did you say at the base they are always pathologic? Where do you hear accidental murmurs?

DR. PREBLE: Anywhere; they may be heard at the apex or at the base, they may radiate all over the chest; they may be very smooth or they may be very rough, very soft or very loud. Of course, in general, you hear them more often over the base than over the apex, and, in general, they are not transmitted. I have heard accidental murmurs so loud that they could be heard through the air, but they are exceptional. You have to differentiate them from the pathologic. Take, for example, an individual who is anemic, a little myocardial enlargement from the anemia, and we get a systolic murmur, and we may have to wait for months to decide whether or not these phenomena are due to changes in the valve or the result of anemia.

INTERN: And after you had waited on the patient like that how could you know then? Would the murmur disappear?

DR. PREBLE: Yes, the murmur would disappear. Do we ever have accidental disastolic murmurs?

INTERN: I don't think so.

DR. PREBLE: I saw a patient years ago at the County Hospital who had a diastolic murmur that could be heard over the sternum, and we argued as to whether it was over the aortic valve or the pulmonary valve. And when he went to autopsy his valves were as sound as anyone's could be. But we do not often find an accidental diastolic murmur. How about the first tone at the apex, would you say that was peculiar?

INTERN: Yes, it seems rather ruffled.

DR. PREBLE: The thing that struck me at once was that I could feel it; it was so sharp. See whether you can hear it.

INTERN: I can't hear it.

DR. PREBLE: I can hear it this far away, 3 or 4 inches. I think if the room was perfectly quiet you could hear it 6 or 8 inches away. You don't hear anything else but that systolic tone; do you get it?

INTERN: Yes, I get it now.

DR. PREBLE: See how far you can follow it. You don't have an opportunity to hear it so well very often.

INTERN: Is it that deep tone? I can hear that 3 or 4 inches away.

DR. PREBLE (to Patient): Do you ever hear that, Philomena?

PATIENT: No, sir.

DR. PREBLE: That's right, as you raise your ear from the chest all the other tones disappear. What lesion is that associated with?

INTERN: I never saw one like that before.

ANOTHER INTERN: I would say a mitral insufficiency.

DR. PREBLE: That is just as sharp a tone or snap as an aortic sound. What lesion is it associated with, Doctor Lewis?

DR. LEWIS: With mitral stenosis.

DR. PREBLE: What is it due to?

INTERN: Due to the heart's contracting upon a small amount of blood.

DR. PREBLE: What is that due to?

INTERN: Due to a mechanical element, to the closure of the mitral valve.

DR. PREBLE: Yes, the closure of the mitral valve; but there are a lot of other factors that enter into it. It is not all from the mitral valve, but the tricuspid enters into it too, and it is possible under certain circumstances that you would get a base tone too. Why, in mitral stenosis, do we get such a loud tone?

INTERN: There is only a small amount of blood for the heart to contract upon and it contracts very well.

DR. PREBLE: It is not the muscular element that is increased here. If you wanted to get a large tone out of a drum what would you do?

DOCTOR: You would hit it.

DR. PREBLE: Why is the tone louder if you hit a drum hard than if you hit it softly? You get a note of the same timbre and get the same pitch out of it whether you hit it hard or softly, but you get a different volume of tone on account of the difference in tension. That is what happens in mitral stenosis. When the heart is beating normally the valve is closed at the end of

systole, but when the valves are not floated up to their normal position they snap up, and it is just like hitting a drum softly or hard. Do you always get this loud tone?

INTERN: I think we always do.

DR. PREBLE: There may be no valvular element at all. Why not?

INTERN: Either because the membrane of the valves or membrane attachments may have too great a tension.

DR. PREBLE: Our membrane is thickened, so that it may be almost like cartilage. Mitral stenosis is never a pure lesion; it is always associated with mitral insufficiency. When we find such a loud first tone at the apex, therefore, we have a right to conclude that we have a mitral stenosis.

Now, we have a good deal of data about it. We have a great big heart, palpable first tone at the apex and palpable thrill at the apex; suppose you outline that heart and see what that will show. Haven't we got enough data now to make a diagnosis?

INTERN: No, I don't think so.

DR. PREBLE: Well, how far can we go in the diagnosis? Have we got enough to say definitely that there is a heart lesion?

INTERN: I think so.

DR. PREBLE: Yes, in spite of the fact that the child knows nothing about it, we have enough to say that she certainly has a heart lesion. Have we got enough to go further and say what kind of a lesion she has?

INTERN: We haven't got all the findings.

DR. PREBLE: Have we got enough to say how much of a lesion it is?

INTERN: We have enough to say we have a mitral lesion.

DR. PREBLE: Yes, a double mitral. What can't we answer, then? Can we make any statements in regard to her aortic valves?

INTERN: Would you say that in all probability there was a lesion of the mitral valve?

DR. PREBLE: Yes, we have enough data now to warrant that diagnosis. Have we any reason to say that the aortic valves are

involved, as occurs so often in mitral lesions? What have we about it that would suggest that?

Doctor: The dislocation of the apex and the capillary pulse.

Dr. Preble: Yes, that's all; only the fact that there is nothing inherently improbable about the child having an aortic lesion. With a child only a light percussion should be used, because otherwise you vitiate your results completely. So you see here we have a cardiac dulness that joins the upper border of the liver dulness $2\frac{1}{2}$ inches to the right of the median line, then goes to the upper border of the third rib on the left, when you percuss lightly. We will look at the orthodiagram and see how it fits with the percussion figure.

Intern: As I remember, it will fit pretty well.

Dr. Preble: What I meant is how closely will the orthodiagram fit?

Intern: I should think it would be larger on account of the angle of divergence of the *x*-rays.

Dr. Preble: Yes, unless the orthodiagram is made at a distance of several feet, it is always larger. This one is nearer than they usually are; corresponds more accurately than they usually do. What is that due to, Doctor (placing a pencil on chest), this increase to the right?

Intern: I don't know.

Dr. Preble: That is the right ventricle, and here is partly the left ventricle and partly the auricle. So we have a generalized enlargement of the heart affecting both the right ventricle and the left ventricle; suppose you listen and find what you can hear. If you want to time it, time it to the carotid. Would you say that child was in good compensation or poor compensation?

Intern: In good compensation.

Dr. Preble: So good that she never has known she has a heart lesion. She can play and dance and run just as hard as anyone, has never had any edema of the feet, no gastro-intestinal disturbance, and yet she must have a lesion of moderate grade on account of the cardiac enlargement, which is considerable. From the intensity of the murmurs one can draw no conclusions.

She didn't know why the school nurse sent her here. What do you hear, Doctor?

INTERN: There is a systolic murmur at the base and one here that ends sharply with a loud tone at the apex. The second pulmonic is markedly accentuated, and at the apex I think I can hear a diastolic murmur.

DR. PREBLE: No, I can't convince myself of that, but you have a loud systolic murmur at the base and accentuation of the tone, but I can't satisfy myself that the child has an aortic insufficiency, although the increase in the size of the heart suggests quite strongly that she might have, but I can't demonstrate it. She has a double mitral lesion, perfectly compensated. What's going to happen to her?

INTERN: I don't think anything will happen to her right away.

DR. PREBLE: What is her greatest danger?

INTERN: The greatest danger is another attack of rheumatism.

DR. PREBLE: The greatest danger is a re-infection of her endocardium, and the next danger is what? If we knew she was going to have rheumatism and not any further involvement of the heart we would say we need not worry about that, but the great danger is infection. What is the next one?

INTERN: I don't know what it is.

DR. PREBLE: What would you say, Doctor?

SECOND INTERN: An infection would make her much worse and she might then get a broken compensation.

DR. PREBLE: Then the next danger is the breaking of compensation. What can we do to avoid these things?

INTERN: It's a question of making her folks understand the condition and let her build herself up.

DR. PREBLE: Except for her heart she is as healthy a little thing as you could know. So far as the re-infection is concerned, I don't know any way we can avoid that. But to avoid a broken compensation? We don't want to make this child a useless little invalid; we must avoid handling her too carefully and yet avoid handling her too carelessly. What can we do?

INTERN: Warn her against extreme exercise.

DR. PREBLE: That's one thing; to guard the child against undue exertion. How much do you run, Philomena?

PATIENT: A little.

DR. PREBLE: You must not do that too hard. Where do you live, on the first floor or up on the third?

PATIENT: First floor.

DR. PREBLE: Are you going to high school, Philomena?

PATIENT: No, sir.

DR. PREBLE: You see this little girl comes from a working family. She will have to work. The family will need some advice as to what they are going to do with that child later on in life. She ought to be better taken care of and have more education, so that she could take up some light occupation, like stenography. If she goes through high school she can secure employment which she can do satisfactorily and go on and live her life comfortably. This child is in sort of a precarious place in her career. She will soon approach puberty, and that is always a trying period for a child, and it has to be especially guarded at that time, particularly if there is a heart lesion. It is wise often to keep a child much in bed or in a chair, for often at that time, even though there is no organic defect, the rapid transition will bring on a heart lesion. Would you give her any medication?

INTERN: Has she a fever?

DR. PREBLE: She hasn't anything. She is here only because the nurse at school noticed that heart condition. We find only a mitral lesion, and she needs no medication. If anything were given it would be either something that was perfectly indefinite or something that was harmful.

SPLENOMYELOGENOUS LEUKEMIA. ADMINISTRATION OF BENZOL, WITH GENERAL IMPROVEMENT

DR. PREBLE: How long has it been, Mrs. Hall, since you entered the hospital?

PATIENT: I came in in February, 1914.

Dr. Preble: Here is the history. This goes back over a year: About nine months before she came in she was troubled with swelling of the feet and ankles, constantly present, but worse at times. There was also swelling in the thumbs. A month later she had some family difficulties and an abortion was induced; she was at that time two months pregnant. Then she had been gradually losing weight, and the feet continued to swell. Shortly after that she noticed that her abdomen was getting large; she had lost in strength and weight and had night-sweats for six months. That is, over a period of about eight or nine months she had indefinite disturbances, loss of strength and energy, and then some months before she was admitted she noticed an enlargement of her abdomen which had reached a considerable degree at the time she came in; that was over a year ago. She has been here almost continually ever since. She looks pretty well, very different from the way she looked on admission. The unusual thing about her, and the reason I am showing her to you, is to point out the difference between her and the patient down in St. Charles' ward, who came in with a history of glandular swelling extending over a period of five or six months.

Now when we look at this patient you see that her abdomen is large and that she has a mass on the left side which reaches out to the median line and passes downward to the crest of the ileum. Suppose you feel that, Doctor, and see what you think it is; can you feel the mass?

Visitor: Yes, plainly; right here I feel it.

Dr. Preble (to Patient): That isn't where you feel it, is it?

Patient: No, sir.

Visitor: Oh, here it is!

Dr. Preble: Yes, and it goes up under the ribs sharply, comes to within a little distance of the median line, is hard, firm, and smooth, and yet the edge is not perfectly even. See how my finger ducks as it goes around in there? I can get my fingers behind it. It is a mass very much longer and wider than it is thick; not very tender. What could that be?

Visitor: An enlarged spleen.

Dr. Preble: Yes, and that spleen under observation has altered much in size; it came down so that you could not get your fingers between the lower border and the pubes and it passed over to the right of the median line. Originally the spleen came over much further. When she came in she had no glandular enlargement that was demonstrable, and she had originally 565,000 whites, of which 3 per cent. were small lymphocytes and 0.6 per cent. large. As the weeks went by the white count fell to 555,000, 495,000, 340,000, 163,000, 50,000, 31,000, 25,000, 11,700, 6500, and that was the last count in May, 1914. That is, through a period of two months the white count was reduced practically 95 per cent. Along with that reduction in whites the reds were 2,100,000 and increased to 2,792,000. That is, she lost the difference between 565,000 and 6500 whites and her red count went up. Then she left the hospital and was out for a little while, and came back in November, and at that time she had 17,800 whites. Then they rose until the count was a little short of 35,000, when she went away again in December. She came back just after Christmas and has been here ever since. The count has been running along between 27,000 and 45,000, but the important thing about her has been not so much the reduction of the whites, but the increase in her reds. They have gone up until she has 4,800,000 with 78 per cent. hemoglobin, so that while there has been a diminution from 600,000 to 40,000 in whites, the reds have gone up from 2,000,000 to 4,000,000 and more, and along with this is the great improvement in her general condition. She is able to do much more and eats well and sleeps well. What is it?

Visitor: A splenic leukemia?

Dr. Preble: Yes; she lay here for a long while, under the influence of benzol, and though the number of white cells was gradually reduced, her general condition did not particularly improve until later. The hemoglobin went up, and while the whites have gone up again to 35,000 or 40,000, that is not so very bad. Her spleen has reduced to half of what it was originally. Now in what way does she differ from the old man down stairs?

VISITOR: She has a chronic splenomyelogenous, and he a chronic lymphatic, leukemia.

DR. PREBLE: How do those two differ essentially; what is the difference between them?

VISITOR: I don't know exactly.

DR. PREBLE: Essentially there probably is not any. In one case we have an enlargement of the glands out of all proportion to that of the spleen, and in the other we have an enlargement of the spleen out of all proportion to the glands. In one case a certain set of blood-building elements are affected to a large degree, and in the other case another set. In all of those cases the tissues are all involved. The acute cases occur every once in a while, and much less frequently do we see a chronic case. When the glands are involved the case is much more likely to run an acute course than when the spleen is involved. Do you know anything about the etiology of it?

VISITOR: Only theories; one is that it is supposed to be due to a toxin, but they don't know anything about it.

DR. PREBLE: That's about right; they don't know anything about it, but there are lots of things about an acute leukemia that suggest an infection. Very frequently they present themselves looking like scurvy, and they suggest to one very strongly that we have to do with an intense, acute infection. That is, they go from acute to the subacute and chronic cases, and they have varying degrees of temperature. This patient has had temperature at various times, but, of course, the existence of the temperature does not necessarily prove that it is an infection. Mrs. Hall has done a good deal better than one might have expected under the administration of benzol.

SYPHILITIC AORTITIS. FACTORS GOVERNING PROGNOSIS. METHOD OF GIVING NEOSALVARSAN IN SUCH CASES

DR. PREBLE: What do you notice about the patient?

DOCTOR: He looks sick.

DR. PREBLE: How do you mean, dark circles under his eyes? He looks all right to me. He has been here less than a week and came in complaining of dyspnea and palpitation with occasional pain in the left side of his chest. The trouble began in October. He is comfortable in the recumbent position and can exercise moderately without bad effect. There is no swelling in the feet. If he is careful he gets along pretty well. What would you think about that for a start?

DOCTOR: I would think it was due to cardiac disturbance of some kind.

DR. PREBLE: What suggests that to you?

DOCTOR: The pain in the chest and the dyspnea.

DR. PREBLE: What about pain in heart lesions?

DOCTOR: He might have an acute endocarditis.

DR. PREBLE: Is pain a common complaint in diseases of the heart?

DOCTOR: Sometimes in aortic lesions.

DR. PREBLE: Are there any other lesions of the heart where it occurs? In cases of myocarditis pain is an average feature, but in the ordinary cases pain isn't a symptom. Do they have pain with pericarditis?

DOCTOR: Sometimes.

DR. PREBLE: Occasionally, but usually they are quite free from pain; here it is rather marked and associated with it is the dyspnea and palpitation, which suggests some sort of a heart lesion. Can dyspnea come from anything else?

DOCTOR: Yes.

DR. PREBLE: How about palpitation?

DOCTOR: Other things may cause palpitation.

DR. PREBLE: Yes, but the combination is suggestive of some disease of the heart. The man says he is in here with broken compensation. Are there any other things about this man that are suggestive?

DOCTOR: I don't remember; he is sitting up in bed.

DR. PREBLE (to Patient): Can you sleep lying down?

PATIENT: Yes, but when I am lying like I am now I am usually more comfortable, but I can sleep in any position.

DR. PREBLE: Is there any other thing about this man that is suggestive to you?

DOCTOR: Nothing except that the history says he is all right if he takes care of himself.

DR. PREBLE: Yes, his compensation is fairly good if he is careful. What is the other thing about this man that is striking? How old are you, Charley?

PATIENT: I will be thirty-six next month.

DR. PREBLE: Doctor, is it of any consequence that he is thirty-five.

DOCTOR: Yes, it would make one suspect an aortic lesion rather than a mitral.

DR. PREBLE: No, that's not the deduction. It is of consequence because of the pain which he has, not because it is an aortic lesion at thirty-five, but because it is *pain* with an aortic lesion at thirty-five. With the presence of pain comes the suggestion that it is on an arterial basis, and what does that suggest to you?

DOCTOR: It might suggest the presence of the spirochete.

DR. PREBLE: Yes, it suggests a syphilitic basis. If he were sixty that suggestion would not strike one so forcibly, but at thirty-five these lesions are most likely to be upon a syphilitic basis. While the dyspnea and palpitation may be readily referred to the heart, the pain suggests that it is an arterial lesion, and that suggests syphilis. Did you ever have syphilis, Charley?

PATIENT: I don't know what that is.

DR. PREBLE: Did you ever have any sores on your penis?

PATIENT: Never but once; I got scalded.

DR. PREBLE: When was that?

PATIENT: About five years ago; but I never had any sores any place on my body, nothing but a few lumps.

DR. PREBLE: Are you married?

PATIENT: Yes, sir.

DR. PREBLE: Have you any children?

PATIENT: No, sir; I've only been married not quite a year.

DR. PREBLE: How about rheumatism?

PATIENT: I used to have it in my fingers, but never very bad.

Dr. Preble: All right, Charley. Let's take off your shirt and see what the chest shows. Now what do you see there?

Doctor: There is a diffuse cardiac impulse.

Dr. Preble: Can you pick out the apex-beat?

Doctor: Yes, I think so; it is right here.

Dr. Preble: In the fifth interspace; you can get a little suggestion of it in the sixth interspace. What else can you see?

Doctor: Pulsation in the neck.

Dr. Preble: Can you see the pulsation in the wrist?

Doctor: Yes, I can see it in the ulnar.

Dr. Preble: Can you see it in the radial?

Doctor: Yes.

Dr. Preble: How about the capillary pulse?

Doctor: Yes, I can see it there.

Dr. Preble: What can you feel here (pointing to base of heart), is that of any consequence?

Doctor: No, I think not; you can get it on a great many people.

Dr. Preble: Yes, it all depends on how high the arch happens to be. Do you feel any thrills over that arch?

Doctor: No, sir.

Dr. Preble: Can you feel anything in particular over the apex-beat?

Doctor: It doesn't seem to be a very strong beat; it feels to me as if it shifted around a little bit.

Dr. Preble: Well, that's what it does, but I wouldn't expect you to be able to feel it. You feel no thrills, no tones?

Doctor: No, sir.

Dr. Preble: See what you can feel up here at the base.

Doctor: There is a little thrill there.

Dr. Preble: A thrill; systolic or diastolic?

Doctor: Systolic.

Dr. Preble: I can't get any thrill there; about the second rib to the left you can get definite pulsation, but one does not feel any thrill there nor any tone. Just percuss that out and see what you can find; don't let yourself be influenced by any marks you may see. Always keep your fingers parallel to the body you are outlining; here is the liver dulness, the superficial

dulness is here, the stomach disturbs it a little bit. It's all right to percuss heavily in a man like this, but in a child you should always percuss lightly. Now this is very interesting, what do you think about that? The heart is increased transversely, so the right border curves slightly inward and crosses the second rib way outside of the sternum, and on the left it is a full $\frac{3}{4}$ inch to the left of the sternum, at the second rib. The cardiac dulness is increased tremendously, and particularly behind the manubrium there is an area $2\frac{1}{2}$ inches wide. What does that suggest?

DOCTOR: An aneurysm.

DR. PREBLE: There is something in there that should not be there, but it is not necessarily of the aorta; it might come from a variety of things. Suppose you listen to that and ascertain what you can hear. Does that strengthen or weaken the suspicion that we might have an arterial process here?

DOCTOR: It strengthens it.

DR. PREBLE: Does it do more than strengthen it? Unless that area behind the manubrium is due to a tumor or some mass, it practically clinches it. We can't say that it is a double lesion yet. Do you hear anything, Doctor?

DOCTOR: I hear a loud, blowing murmur there.

DR. PREBLE: Is it systolic or diastolic?

DOCTOR: Diastolic.

DR. PREBLE: Anything else? You know when you listen to the heart you should always listen to the tones and get them fixed in your mind and then listen for abnormal symptoms.

DOCTOR: The murmur is such that you can't get any tone.

DR. PREBLE: Can't get any?

DOCTOR: Perhaps one tone.

DR. PREBLE: Which one?

DOCTOR: The systolic.

DR. PREBLE: Yes, the systolic; the diastolic is replaced by the murmur. Right over the base you can hear the systolic and diastolic murmurs and one can hear also two tones at the apex. There is one that comes up from the base, but the diastolic element becomes much more prominent with a long-drawn,

blowing sound, with the point of maximum intensity in the second intercostal space to the left. The second pulmonic tone can be heard, but the second aortic is replaced by the murmur. Can you hear that?

DOCTOR: Yes, sir. I get the diastolic murmur all over the sternum and can trace it down to the apex.

DR. PREBLE: What would you think about the etiology of the thing?

DOCTOR: I should say that without a doubt it was specific, from the fact that he is so young and that it has affected the aorta before the valves. What was the Wassermann?

DR. PREBLE: I can't report on that yet. At least the man has never been under any treatment for luetic infection, but the age and the localization and the aortitis make it practically certain. What about the heart? If he takes care of himself and doesn't do too much he gets along all right. The point of the question is this: Would you put that man on vigorous antisyphilitic treatment?

DOCTOR: Yes, I would.

DR. PREBLE: What good will it do if he has a lot of scar tissue there?

DOCTOR: I wouldn't say that it was an old affair, he has had these symptoms only six or eight months.

DR. PREBLE: Probably; I don't believe that anybody would argue with you about the advisability of putting him on antisyphilitic treatment. The thing suggests strongly that the lesion is permanent, but still one is often deceived about these things. I remember a colored girl who came into the hospital with a story which would lead you to infer that if she had syphilis it must be of years' standing and the physical findings were those of an aortic insufficiency or an aortic aneurysm, and it was assumed that the syphilitic changes were old chronic changes. She had not been in the hospital many days when all of a sudden she passed away, and the autopsy showed that the changes in her aorta were not chronic, but were acute, and the girl had not been telling us the truth about her history. If we had put that girl on vigorous antisyphilitic treatment and could have gotten it

introduced earlier, it is quite possible the girl could have been pulled through, and I think this man ought to be put on vigorous antisyphilitic treatment. What would you do for him in the way of treatment? There are two problems that present themselves: How much treatment does this unstable compensation require and how much shall we do for his syphilis?

Doctor, how much does that heart need in the way of cardiac medication?

DOCTOR: I don't think he needs anything now.

DR. PREBLE: I don't either; I think rest will be sufficient to bring about compensation. How about the other element?

DOCTOR: I think we have a clear indication to treat him specifically.

DR. PREBLE: How about the arsenical preparations, would you give them intravenously or intramuscularly?

DOCTOR: I would give them intravenously.

DR. PREBLE: Yes, but instead of giving him a large dose of neosalvarsan, give him repeated small doses, because with the question of this being an aortitis it might not be wise to give large doses of so active an ingredient. Give 1.10 m.m. at a dose, and then in a few days give him another one, rather than give the ordinary sized dose. At the same time make use of mercury; give him some inunctions.

Let's get a look at you in the *x*-ray room, Charley.

The fluoroscopic examination shows shadows corresponding in size to the areas outlined by percussion. The prognosis in cases of this sort is unfavorable because of the constant danger of sudden death. Death takes place through the same mechanism as in angina. In addition to this unfavorable element in the prognosis, they are always likely to have a break in the compensation severe enough to be dangerous; and still other syphilitic manifestations throughout the body, particularly in the nervous system, may develop. However, unfavorable as this sounds, many of the cases under the influence of antisyphilitic treatment undergo great improvement, and may for years be entirely free from anything that suggests that they ever had a syphilitic aortitis.

CLINIC OF DR. MAURICE L. GOODKIND

MICHAEL REESE HOSPITAL

SPLENIC ENLARGEMENT—A CARDINAL SYMPTOM OF VARIOUS DISEASES

TODAY we shall discuss diseases in which we find enlargement of the spleen the dominant feature in the clinical picture of our patients.

Usually, under normal conditions, one is not able to palpate the spleen, and on percussion the spleen is found in its vertical diameter to be from 4 to 7 cm.; but there are certain factors which frequently prevent a proper determination of the size of the spleen by percussion, such as an inflated or distended stomach (this factor we can somewhat overcome by having our patient lie upon the right side), a distention of the colon at its splenic flexure with gas or with fecal matter, a high diaphragm as a result of meteorism and ascites; so that under ordinary conditions the most reliable method for determining the enlargement of the spleen is through palpation.

We have the spleen enlarged in various acute infectious diseases, notably, typhoid fever and typhus fever; enlargement sometimes occurs in children with high temperatures and enlarged glands (Still's disease), in rickets, in syphilis, in malaria, in diseases of the liver, especially the cirrhoses, and in amyloid disease; in cancer of the liver it is rather the exception than the rule to have an associated splenic enlargement. There are certain conditions in which primary enlargement of the spleen occurs: splenomegalia of the Banti type, of the Minkoski type, of the Gaucher type, and the various types of leukemia and pseudoleukemia.

Case I.—The first case we shall demonstrate today is a man forty-eight years old, shoemaker by trade, Roumanian. He was in the hospital four years and also three years ago. He returns to the hospital complaining of symptoms similar to those which he had at previous times.

To summarize briefly, his salient symptoms are pain in the epigastrium independent of food, exertion, or weather conditions; marked constipation, headache, and dizziness. He has apparently not lost in weight. His family first noticed the change in his complexion; that his face was assuming a markedly brick-red color, and this was more particularly noted during the cold weather. Outside of that the man has had no further complaint.

As we examine him we find a well-nourished man, with no special peculiarities in appearance other than this markedly brick-red color of his skin. When he lowers his hands the tips of the fingers and the finger-nails assume an intensely cyanotic hue, and on elevation of the hands we find they become markedly white.

Many of these patients manifest other vasomotor changes of the skin—dermography, erythromelalgia. Sometimes a condition is present analogous to Raynaud's disease. In others we may have an angioneurotic edema (Quincke's disease).

This man's systolic blood-pressure is 160, diastolic 100, pulse pressure 60 on the right side, and practically the same on the left. His blood-picture was very interesting: We find an enormous increase in the number of red blood-corpuscles, polyglobulism (8,000,000 to the cubic millimeter); but there are no other changes in the erythrocytes (no nucleation, megalocytes, or poikilocytes). The whites are not increased in number, but the hemoglobin, taken by Sahli, also by Fleischl, was 140.

The physical signs, other than this very marked red hue of the skin, consist of a very extensive enlargement of the spleen. It extends 10 cm. below the left costal margin.

Let us then group the symptoms which we have elicited in this man: We have polycythemia, a high hemoglobin, a very marked cyanosis of the skin, especially of the face, and an enlarged spleen.

This man's blood we have very carefully examined spectroscopically in order to rule out sulphemoglobinemia and methemoglobinemia, conditions which may arise as a result of hemolysis of the blood from the ingestion of the various coal-tar products (phenacetin, veronal, sulphonal) from CO_2, $KClO_3$, as well as poisons which may be generated within the intestinal canal (enterogenous cyanosis).

We have, then, a well-marked case of a condition that was first described by Vaquez and afterward closely studied by Osler. The condition is probably due to some toxic agent generated within the intestinal canal which causes the changes in the spleen, the dusky hue of the skin, and the polycythemia. It is known as Vaquez's disease.

These cases occur much more frequently in the male subject, usually between the ages of thirty and fifty. There is no specific treatment for this disease. When the blood-pressure becomes very high and evidences of cerebral congestion arise a venesection is frequently of service, abstracting 300 to 500 c.c. of blood. We also give them an occasional nocturnal dose of blue mass followed by some aperient water.

The diet should not exceed 2000 calories a day. Alcoholic beverages and foods rich in purins should be excluded.

Case II.—John T., a Russian, thirty-nine years of age, was perfectly well up to the middle of January. At that time he sustained a blow on the back of the neck. He was confined to bed with pain in the lumbar region and in the shoulders for several weeks. Since then he has had numerous eructations of a tasteless character, feeling of great weakness, and loss of weight. Also complete revulsion to food; practically everything he eats nauseates him. During these several months he has lost 30 pounds. He has also considerable abdominal distention and some epigastric pain, which is increased by eating and by walking. About seven weeks ago he had two or three slight nosebleeds and also noticed spots appearing on his body—vibices and petechiæ.

He has had no pulmonary, cardiac, or urinary disturbance.

His main symptom at the time he entered the hospital was this progressive weakness.

He denies alcoholic or venereal history, or any previous illness.

As we examine this patient we find him in a condition of greatly impaired nutrition, the skin is dry, he exhibits very peculiar pigment marks which do not disappear on pressure, varying in size from a pin-point to the palm of the hand. These pigment spots are of a brownish color and are distributed over the entire trunk and extremities, as well as appearing on the mucous membrane of this man's mouth. In addition to these pigment spots within the mouth we notice a very pronounced pyorrhea.

On examination, we find the lungs normal. Heart somewhat enlarged toward the right ($10\frac{1}{2}$ cm. to left, 7 cm. to right of mid-sternal line). A murmur is present over the mitral area, of a soft blowing character, and in all likelihood is due to relaxation of the heart muscle.

Examination of the blood: 3,500,000 red; 80 per cent. hemoglobin; 14,000 white, of which 75 per cent. are polynuclear neutrophils.

The blood-pressure examination yields a very interesting result: We have systolic blood-pressure of 80, right arm, with a diastolic of 55, which is an exceedingly low blood-pressure for a man of this age and weight, and is indicative of a rather serious circulatory disturbance, due, in all likelihood, to an interference with the function of the adrenals.

The urine contains free bile-pigment, but in other respects is quite normal.

The stomach analysis: A total acidity of 40, free HCl 20, no blood, no lactic acid, no Boas-Oppler bacillus.

The liver extends from the fifth intercostal space to a line almost on a level with the umbilicus. In fact, we can see a very marked prominence in this area. On palpation we meet with fairly firm resistance, no special pain or tenderness, with a round border and smooth surface, without nodules.

In the left hypochondrium we find an organ the surface of which is harder than that of the liver, extending 4 cm. beneath the costal margin, with a very distinct incisura or notch; it moves

freely with respiration, extending upward to the sixth intercostal space. This organ is the spleen.

We have, then, in this patient: Enlarged liver and an enormously enlarged spleen, with brown pigment spots in the skin, and extremely low blood-pressure. The urine contains free bile, but the stools are not acholic. He has neither ascites, jaundice, nor varicosities. He has no alcoholic history. The Wassermann was negative. The blood examination is that of a simple anemia. This symptom-complex constitutes a disease which we call hypertrophic cirrhosis of the liver of the type of Hanot.

We may have intense icterus in this condition which may be persistent, or it may come on in separate attacks lasting from a few days to a few weeks, associated with high temperature and marked gastro-intestinal disturbance. Hemorrhages may occur from the various emunctories, but are not always present. The disease is an insidious one and may last for many years. It is due to some unknown form of intoxication.

The treatment is purely symptomatic. The *x*-ray and arsenic do not yield specially satisfactory results in this type of splenic enlargement. On account of this man's low blood-pressure and pigmentation of the skin, both evidences of the dysfunction of the adrenals, we shall give him epinephrin, 10 minims of a 1 : 1000 solution, hypodermically, three times a day, and place him on a nourishing, easily assimilated diet.

While the prognosis as to life is good, this disease tends to last many years. I do not know of a case in which a natural cure has occurred.

Case III.—From the multitude of names given to the next example of splenic enlargement, which we are about to demonstrate to you, conclusions may be drawn of the chaotic and ill-defined position in literature which this disease has occupied in the past, but today, thanks to the labors of various investigators, it has been rescued from this confusion of nomenclature, and it is now regarded as a distinct entity, both clinically and histologically.

Our patient is a young man, seventeen years of age, Polish

nativity, clerk by occupation; he has a perfectly negative family and personal history previous to the onset of his present illness, which began rather insidiously six months before his entrance to the hospital (about September, 1914), with gradual loss of strength and weight, increasing pallor, occasional darting pains in the arms and legs, and periods of high fever which would last for several weeks, terminating by crisis, followed by complete remissions for several weeks. These cycles of alternating attacks of febrile movements and freedom from temperature would occur rhythmically at intervals of two weeks, in this feature bearing close resemblance to the febris recurrens (Spirochæta spirillum of Obermeier). One of the first objective phenomena to which his attention was drawn was the gradual enlargement in the right side of his neck. After persisting several months in this locality, it became apparent on the left side. But at no time during this period has he had any difficulty in deglutition, throat inflammation, or involvement of the gums or teeth.

Following these enlargements, which were first noticed in the neck region, he began to have enlargement in the left hypochondrium, which caused him considerable discomfort because of the dragging sensation in that neighborhood. He also noticed enlargements in both groins. He began to experience sensations of lassitude and weakness, as before described.

On physical examination we find an exceedingly cachectic and anemic appearing lad, with no skin changes apparent. His pulse was 96, regular, small. Systolic blood-pressure 106, diastolic 80, pulse-pressure 26. His cardiac area could not be well defined because of irregular areas of dulness radiating from either side of the sternum in the third and fourth intercostal spaces, which on fluoroscopic examination proved to be enlarged mediastinal glands. No abnormal sounds, murmurs, or reduplications could be determined.

The spleen extends 5 cm. below the left costal margin and is hard, but not nodular. No physical changes could be made out in the lungs, liver, or nervous system.

The superficial lymph-nodes in the neck, both the anterior and posterior cervical group, are enlarged and vary in size from

a hazelnut to a walnut. They are elastic, firm, but not painful to touch, distinctly encapsulated, and do not tend to fuse, nor does the skin above them pucker nor is it adherent. These masses pass downward underneath the clavicle and sternum. The inguinal and epitrochlear glands are similarly involved.

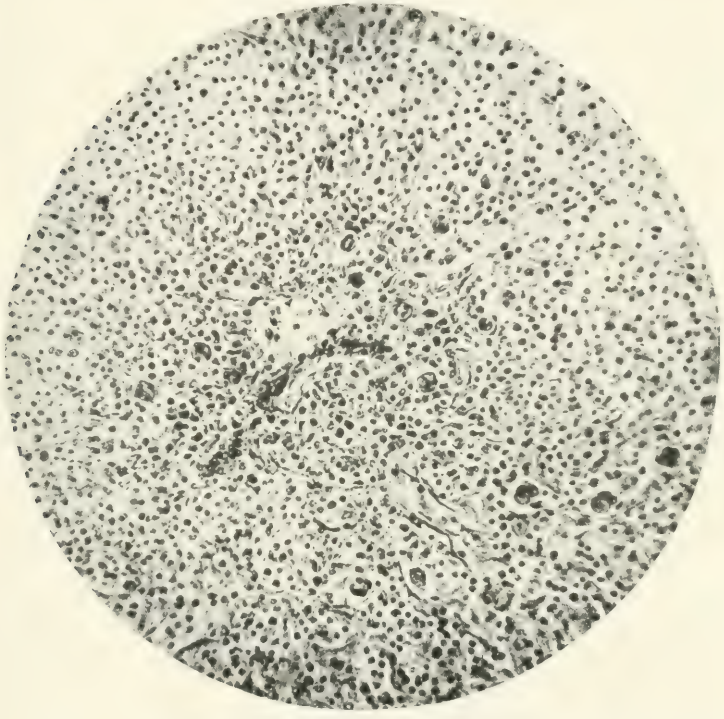

Fig. 77.—Detail from lymph-node (215 diam.). Note numerous endothelial giant cells and fine fibrillation. Also obliteration of normal architecture of node by general cellular hyperplasia. The numerous eosinophiles are not differentiated in the monotint.

There are no evidences of intra-abdominal glandular enlargement —negative rectal examination, absence of venous engorgement and ascites, no disturbance of visceral function. A section of one of these glands excised from the neck shows very characteristic changes: (1) Giant cells, the greater part of which are uninuclear, although some are multinuclear, containing four nuclei;

(2) eosinophile cells; (3) a marked proliferation of the connective-tissue stroma and endothelial cells forming a reticular mesh

Fig. 78.—Skiagram showing stomach filled with bismuth demonstrating enlarged splenic shadow.

(granulation tissue), a histologic picture best described by Dr. Dorothy Reed.

Fig. 79.—Skiagram demonstrating cervical, mediastinal, and peribronchial glands: *a*, Mediastinal glands; *b*, cervical glands. Note apparent enlarged aortic and cardiac shadow caused by enlarged mediastinal glands.

A bacteriologic examination in this connection failed to reveal diphtheroid organisms, first described by Negri and afterward by Bunting and Yates. Parenthetically I may state that this

organism is no longer recognized as peculiar to this disease, for it has been found in all forms of glandular swellings and is absent in many cases of typical Hodgkins.

Laboratory findings: The blood, hemoglobin 52 per cent., red blood cells 4,224,000 (no nucleated or distorted or misshapen red blood cells), white blood count 12,000, of which 80 per cent. are polymorphonuclear neutrophils, 19 per cent. mononuclear, 1 per cent. transitional, no eosinophiles. Repeated blood-cultures were negative. The Wassermann of the blood and spinal fluid were negative. The feces were alkaline plus bile, no blood, ova, or mucus. Pancreatic functional tests negative. The urine was 1028, acid, free from albumin, sugar, acetone, albumose, and casts.

Tuberculin tests (percutaneous and von Pirquet) were completely negative.

x-Ray examination demonstrated an irregular mediastinal field, and by filling the stomach an enlarged spleen became evident.

Then, grouping together the blood findings, the clinical history, physical signs, and histologic picture, we shall have no difficulty in diagnosing the disease from which this young lad is suffering as a typical Hodgkins.

Formerly all glandular enlargements with splenic changes were thus grouped, whether they were lymphosarcomas, tubercular or syphilitic adenopathy, pseudoleukemias of the von Jaksch and other types, or various types of splenomegalia (Banti, Vaquez, Minkoski, and Mikulicz), but today we have certain criteria, all present in this patient, which permit us to make an absolute diagnosis of the disease.

The treatment consists of the continued administration of the various preparations of arsenic. This may be given by the mouth in the form of Fowler's solution in increased dosage, or more efficaciously given hypodermically as atoxyl or arsacetin, or cacodylate of soda; while the hypodermic administration of arsenic is more efficacious than when given per orum, and is less apt to produce gastro-intestinal derangement and pigmentation of the skin, on the other hand, there is greater danger of

producing neuritis, especially of the eye nerves, through this method.

In addition to the arsenic treatment, we administer to the glands and over the spleen systematic applications of the Röntgen ray. In a number of cases thus treated (in conjunction, as a matter of course, with hygienic and dietetic measures) very successful results have been achieved.

The removal of the spleen in this form of splenic enlargement has not been of any benefit. The same thing may be said of the practice which is at present in vogue in pernicious anemia. In fact, the only form of splenomegalia in which removal of the spleen has been of any decided value has been in Banti's disease.

CLINIC OF DR. RALPH C. HAMILL

COOK COUNTY HOSPITAL

TWO CASES OF LOCOMOTOR ATAXIA

DR. HAMILL: In the last clinic I showed you examples of vascular and perivascular forms of syphilis. You will remember in the scheme that I gave you I spoke of the two main divisions, namely, the vascular and perivascular and the parenchymatous. Today I wish to show examples typifying the parenchymatous forms.

Of the parenchymatous forms I will show you 2 cases of locomotor ataxia or tabes dorsalis, and 1 case of primary optic atrophy. This first patient, W. W., is thirty-three years of age, a waiter by occupation. At twenty he acquired a chancre. About two weeks later there was an eruption spread over the skin of the body. He took pills for two months for this trouble. He has been married for five years, his wife is living and well; there was one miscarriage at about three months, no other pregnancy. There was no suggestion, no symptom of the presence of the disease for about eight years. Then he had a peculiar symptom which may or may not have been due to the original infection. A flash of light, momentary in duration, would come in the extreme temporal portion of the right field of vision. It seemed to be about the size of a pea, would come four or five times a day; for periods it might occur daily, at other times he might be free for a week or two. This continued for a period of a year; the vision was not affected thereby. There was no dizziness or headache associated with the flashes.

Although at first questioning this seems to be the first symptom of the return of the disease, subsequent direct questioning brings out the fact that about a year before the appearance of

these flashes he began to have slight pains above and below the knees. These he speaks of when asked "Did you have rheumatism at any time within the past six or seven years?" He answers, "Oh, yes, four or five years ago I had rheumatism," and then goes on to describe these stabbing pains recurring every five to ten minutes over an hour or two with periods of freedom lasting as long as a month. It appears somewhat characteristic of the pains that they were more apt to come if he was tired. As I say, they were repeated jabs in a spot about the size of a dollar, and during the period of active pains the spot would be quite sore, but as soon as the pains ceased the soreness disappeared. These pains have gradually become more frequent. This morning he is suffering from an attack of similar pains running from just above to just below the left ankle along the front of the leg.

DR. HAMILL (to Patient): Is it sore there (touching the shin)?

PATIENT: Yes; I can hardly touch it, and it is so sore I can't stand on it.

DR. HAMILL: You see there is no redness of this area, which is manifestly tender, as shown by the way he lifts the sock off the skin. These pains are the characteristic shooting or stabbing pains of locomotor ataxia; probably the most frequent cause in this disease which leads patients to seek the advice of physicians. They can stand some disturbance in locomotion, some difficulty in getting about in the dark and not think very much about it. They will even frequently bear considerable disturbance in the function of the bladder, either a slight incontinence or, more commonly, a hypesthesia or anesthesia of the bladder mucosa, which allows them to go all day without passing the urine. Or they may notice that their vision is failing, putting up with this for perhaps a year or two without seeking advice. The pains, however, recurring time after time, lasting over periods of from one to twenty-four hours, almost always drive them to seek relief.

I have already given you a description of the characteristics of these pains. Their occurrence in the neighborhood of joints, as with this man now about the ankle-joint, formerly about the

knees, naturally leads the sufferers to consider the pains as rheumatic. This belief of theirs is borne out by the fact, as this patient states now, that standing on their feet or any movement of the affected parts is practically impossible. The belief in the rheumatic character of these pains is also borne out in many patients by the fact that they are frequently very much relieved by doses of from 10 to 15 grains of aspirin, a remedy, in the mind of the lay public, for rheumatism. I dwell upon the nature of these pains because they so frequently precede the ataxic stage of locomotor ataxia, because they are so characteristic, and because it is possible by intensive, vigorous treatment, when put upon the right track by these pains, to prevent the further progress of the disease in many cases.

In this particular case the pains preceded any difficulty with his gait by over four years. About two years ago he began to have trouble controlling the bladder, especially if he took a little too much liquor. This grew gradually worse until five months ago, at which time he could not feel the passage of the urine; had no idea when he passed it. Now he has a warning, but has to hurry to avoid wetting himself.

The first disturbance with locomotion was observed about a year ago, when he found that he could not run easily. About eight months ago he gradually became apprehensive about his walking. This was especially noticed in the daytime when he was walking on a tile floor; he was fearful of slipping if he did not step carefully. He does not remember that walking in the dark or going down stairs inconvenienced him.

Also eight or nine months ago he first noticed a binding sensation at the level of the belt—"It felt as though my pants were too tight." Since coming into the hospital on the 6th of January the disturbance with the gait has gradually become more marked, so that now he presents a perfectly typical picture of the tabetic gait. As you see, when he attempts to rise from his chair his legs shoot out, are brought down to the floor with a stamp, and he rises to his feet, as it were, in sections—by jerks. He stands with his feet well apart, the knees slightly hyperextended and curved backward. As he starts to walk his feet are rather far

apart, they are thrown rather violently, overreaching the full limit of the step, which ends in a pulling back of the feet and the setting them down with a stamp. You notice another characteristic: Instead of carrying his weight between his two legs as they are spread at the completion of the step, and rising on the toes of the rear foot, he seems to carry his weight entirely on the rear foot until the moment when the forward foot has been put flat on the floor, then he draws his body weight on to this forward foot. Instead of his gait being propulsive, it is tractive. You see it is impossible for him either to stand or walk with his eyes closed. It is evidently necessary for him to maintain his balance through the corrective influence of and the information derived through his eyes; that is, there is not sufficient information conveyed to the motor centers of the position and degrees of tension of the various muscles used in gait and station to allow of reflex, involuntary stability.

This same defect is the cause of another symptom or sign commonly referred to as the "heel-knee" when demonstrated in the leg, and the "finger-to-nose" when demonstrated in the arm. As he lies on his bed and tries to put the right heel on to the left knee he does it quite well with his eyes open, but with the eyes closed there is considerable excess movement. He lifts the leg too high and he settles it on to the knee too sharply. The finger-to-nose test is relatively well performed, but you notice that as the arm comes in slowly, approaching the nose with the forefinger, there is a swaying—the tracing of an irregularly curved line rather than the straight line a normal individual would make.

Further examination reveals a slight irregularity in the shape of the pupils, a tendency toward oval. The right is distinctly larger than the left. There is practically no reaction of either pupil to light, whereas the reaction to convergence is prompt; that is, when one asks him to look out of the window and then at one's finger held 6 inches from his face, the pupils come down sharply. There is no nystagmus. The fields are not grossly contracted. There is a slight cupping of the disks with a suggestion of beginning optic atrophy. The tongue shows a coarse tremor. There are many palpable glands in the posterior cervical

triangles, as well as in the groin. The heart and lungs are normal. There is nothing pathologic in the abdomen.

The deep reflexes in the arms are distinctly diminished; the knee jerk and the Achilles jerk are lost; stroking the foot sole causes a frank plantar flexion of the toes; the cremasteric reflex is gone; the abdominal, brisk. There is complete loss of sensation of movement in the joints of the foot, knees, and hips; that is, any change in the position of the leg fails to register so long as his eyes are closed. He loses entirely knowledge of its position as soon as he closes his eyes. You see he names correctly even very small changes of position in the fingers; the sense of position seems practically normal.

There is a diminution of pain and touch sense of the entire trunk, of the ulnar side of the arms, and of the legs; that is, although he frequently (in fact, usually) is able to say whether the point or the head of a pin is used in touching him, still there is no wincing, no particular expression of pain elicited by repeated jabs of the pin in one place.

A very characteristic finding, however, is the one I now show you as I grasp his Achilles tendon between the knuckle of my thumb and the forefinger. As I squeeze hard, using all my strength, he gives no evidence of pain. If you will try it on yourself you will find that it is a very unpleasant sensation even when only slight pressure is applied. The same is true of the testicles. Also when I roll the ulnar nerve in the notch between the olecranon and internal condyle it gives only a tingling feeling in the little finger; no pain at the point of pressure. These three signs are frequently demonstrable in early tabes when other disturbances of sensibility are so slight as to be practically unappreciable. For this reason they should always be looked for.

The urine is normal; blood-pressure is normal. A lumbar puncture on the 27th of January showed twenty-five cells to the cubic millimeter and a positive Nonne reaction; that is, an increase in the globulin content. A second puncture on the 9th of February showed sixteen cells to the cubic millimeter and a strongly positive Nonne.

Case II was admitted to the hospital July 1, 1913. He is

forty-five years of age, a clerk by occupation. He had a sore on the penis, called a soft chancre, twenty-two years ago. It was followed by no skin eruption, no sore throat, no sores in the mouth, and no headache. He took no medicine. He was free from any signs of the disease for twelve years. Then, ten years ago, he commenced to have sharp, shooting pains in the bone and in the fleshy part of the thigh. Each pain was of momentary duration; the pains were frequently repeated for about half an hour, then he might be free for as long as a month. The same description applies to his pains as to those of the first case. They gradually became more frequent and more constant.

About eight years ago he had an attack of double vision lasting but two or three days. He thinks the left eye turned in. As with the former patient no mention was made of his rheumatic pains until direct questioning was addressed to the subject, so with this patient no mention was made of this very short period of double vision until he was questioned directly concerning its possible occurrence. The double vision came on without headache, but with slight dizziness. It passed off without taking medicine.

Six years ago he commenced to have terrible pain in the stomach. The first attack lasted for about ten days, during which time there were almost constant cramps at night. The taking of food made no difference and there was no vomiting. Ever since then he has been constipated and has had a sensation as if he were wearing too tight a belt.

Four or five years ago he began to have less and less desire to urinate, the quantity seemed diminished; the sensory power diminished at the same time. Also at that time he commenced to have a numbness in the soles of the feet. The gait became uncertain, so that he had to keep his eyes on the ground, and it became more and more impossible for him to walk in the dark. Walking down stairs also proved more and more difficult.

Two years ago he got up one morning, stood on his feet, his legs gave out suddenly, and he was unable to walk at all. This was after he had been using a good deal of alcohol for several years.

After his admission to the hospital, and he had spent five days in bed, he was able to walk fairly well. He walked about the wards until three months ago, when he commenced to get worse again, and for the past three weeks he has been unable to walk without assistance.

On examination, you see he is a rather thin, decidedly sallow individual. Any movements of the legs demonstrate a very marked ataxia. As he tries to cross one knee over the other you see the leg is thrown out at right angles to the trunk, jerked over the other knee, and the foot is dropped, regardless of the fact that he cracks his heel rather sharply against the chair.

The pupils both react to light, but sluggishly; that is, when shut from daylight and then the light is suddenly allowed to shine into them they come down with appreciable sluggishness. Their reaction to convergence is much sharper. Ocular movements are normal and the fundus is normal. At least there is no gross change in the head of the optic nerve.

The plantar reflexes are normal, the cremasteric is lost, the abdominals are brisk; the Achilles, patellar, wrist, biceps, and triceps reflexes are all lost. Sensation of change of position is fairly well retained in the fingers and arms, but is entirely lost in the legs and feet. A pin prick is usually called sharp on the trunk, but is said not to be painful. On the other hand, scratching any place on the body causes a complaint of pain after a scratching of 1 or 2 inches, but there is a marked perseverance of pain; that is, he continues to complain of the pain as an active sensation for five or ten seconds after being scratched. He shows the same loss of deep pain sense in the Achilles tendon, the testicles, and the ulnar nerve that the first case did.

The urine shows a trace of albumin. The heart and blood-pressure are normal. A lumbar puncture done this morning shows forty-nine lymphocytes to the cubic millimeter, with positive tests for globulin.

There is no question of diagnosis in either of these cases. The disturbance of gait is so typical as practically to make the diagnosis itself. There is but one other condition in which we get a similar disturbance, and that is a combined degeneration in the

spinal cord, as is found commonly associated with pernicious anemia. In combined degeneration there is practically always a Babinski sign—stroking the sole causes an extensor erection of the great toe instead of the normal plantar. This is a sign, probably the most reliable, of involvement of the pyramidal tracts, something which does not occur in locomotor ataxia. In combined degeneration we do not find an increase of cells in the spinal fluid; in combined degeneration we do not find the pupillary reaction seen in this case typical of late syphilis, namely, good response to convergence, diminished or lost to light.

The only other disease which can at all simulate this disease picture is peripheral neuritis, occurring usually in alcoholics and commonly called alcoholic neuritis. In peripheral neuritis we can get the same losses of sensation, the same losses of reflexes and pains, which are somewhat similar. There is this difference, however, in the sensory findings of peripheral neuritis: whereas in locomotor ataxia deep sensation, such as that of the Achilles tendon, is diminished or lost, in neuritis it is almost safe to say it is increased. Any pressure whatsoever on the muscles and tendons of the lower legs is apt to be bitterly complained of. Also, the pains of peripheral neuritis differ from those of locomotor ataxia in that they are much more constant, and that there is frequently a dull, heavy pain associated with the sharp, stabbing pains.

Where we have pupillary changes in peripheral neuritis we find a paralysis or loss of reflexes to both light and convergence instead of only to light, as in locomotor ataxia. It is very rare, indeed, to find any increase in the cell contents of the spinal fluid in peripheral neuritis. The characteristic motor findings of peripheral neuritis also assist in differentiation. Here we get true paralyses; notably the foot-drop, entire inability to flex the foot dorsally, or a paralysis of the anterior tibial group of muscles.

In the explanation of the pathogenesis of the symptoms of these two cases we will consider first the pains. The most widely accepted explanation conceives a very chronic meningitic process involving the posterior roots and posterior ganglia. Both by mechanical irritation and by direct toxic action this meningitic

process irritating the posterior or sensory nerve-roots causes pain. This idea of a meningitis is very well borne out by the findings in the spinal fluid, namely, increased cell and globulin content.

According to Nagotte, this meningitic process about the posterior roots close to their point of entrance into the spinal cord is the essential lesion of locomotor ataxia. The root-fibers are here caught in the chronic inflammatory process and secondary degeneration is established. This shows itself typically in the degnerative changes in the posterior tracts of the spinal cord. Through these tracts run the fibers of joint and tendon sensation, some of the fibers of touch, and, for a very short course, the reflex fibers. The fibers carrying the sensations of pain probably pass through the posterior tracts along the border of the posterior horn, or else they are involved only at the point where they enter the spinal cord. The finding of spirochetes in the posterior columns of the cord may explain in part why in many cases their involvement is so much more apparent than that of the tracts conveying pain.

The prognosis in these two cases is bad, so far as any particular recovery is concerned. One of them has had 192 hypodermic injections of sodium cacodylate without any influence on his symptoms. The other, the first patient, has had two intraspinous and three intravenous injections of neosalvarsan without any particular improvement.

It is safe to say that mercurial or potassium iodid treatment would have been equally ineffective. However, because of the changing of services it has been difficult to give constant treatment, and perhaps treatment has not had a fair show. The consensus of opinion, however, is that in parenchymatous syphilis antisyphilitic treatment does not have very much effect. If it is, as Nagotte has said, true that the pains are directly due to a meningitic process, then we can explain why in some cases vigorous antiluetic treatment seems to influence them in a decidedly beneficial manner.

A CASE OF PRIMARY OPTIC ATROPHY

Dr. Hamill: This third patient is thirty-nine years of age, a motorman by occupation. He came into the hospital because of failure of vision, which began about two years ago, with rapid loss during August and September, 1914. The disturbance began in the left eye. Along with this visual disturbance there has been some slowness in starting micturition for the past two years, so that now he can go practically indefinitely, as he says, without urinating, and when he attempts to pass water he may have to wait one-half to three-quarters of a minute before getting it started. He has felt no desire to urinate and has not emptied his bladder since 9 o'clock last night; that is a stretch of fifteen hours. Pressure about the symphysis, as you see, is somewhat distressing and he says causes a desire to urinate. Another factor of note is brought out by observing the flow: half a minute is required for its start, and then its passage varies from moment to moment; that is, there will be free flow for a few seconds, then almost a complete cessation, and then a flow again, and so on.

Accompanying this difficulty there is some diminution of sexual power; not enough, however, to be of any great consequence.

Last August he had two or three attacks of severe pain in the left shin bone. This pain was somewhat intermittent.

Dr. Hamill (to Patient): How long did it last?

Patient: It would last three or four minutes, and perhaps would return in two or three nights.

Dr. Hamill: This occurred only two or three times. There have been no other subjective sensory disturbances.

In the examination, the right pupil is found to be definitely larger than the left. There is practically no reaction to light, whereas there is good reaction to convergence in both eyes. This pupillary change is that which is found in the late syphilitic pupils, namely, the Argyll-Robertson pupil. The disks, described by Dr. E. V. L. Brown, show a primary optic atrophy; that is, they are described by him as bluish white, slightly cupped, the vessels much narrowed, and the outlines sharp. In the right eye vision equals $\frac{11}{200}$. In the left he sees shadows only.

There is a slight blunting of pain sense on the lower legs; when he is repeatedly stuck with a sharp pin point about every fifth or sixth time he calls the point dull instead of sharp, a thing a normal person would never do.

The plantar reflexes are normal; there is a bilateral foot-clonus, and suggestion of a knee-clonus on the left side; the deep reflexes in the arms are normal. The abdominals are present and apparently normal; the jaw-jerk is absent. In spite of these reflexes, indicative of spasticity in the legs, he has experienced no trouble with the gait.

This disease has come on without headache, vomiting, dizziness, or symptoms of increased cranial pressure found in 70 to 80 per cent. of brain tumor cases. There is no history to suggest poisoning of any sort. I speak of this because primary optic atrophy is sometimes induced by such extraneous poisons as methyl alcohol and arsenic.

The causes of primary optic atrophy are not many: syphilis of the nervous system, multiple sclerosis, arteriosclerosis, combined degeneration of the spinal cord, brain tumor, and the poisons I have mentioned being the chief ones. Of these, syphilis is very much the most frequent.

This patient has had numerous venereal infections, although in only one did he have anything very suggestive of syphilitic origin. This was about fifteen years ago, when he had what sounds like a condyloma on the head of the penis. He has had no secondaries, no eruptions, no sores in the mouth, or anything that suggests syphilis.

In the differential diagnosis of these cases each smallest sign must be considered. Perhaps the most instructive fact in connection with the case is the evidence of lesions or disturbances in definite parts of the central nervous system. Primary optic atrophy implies disease of the optic nerves themselves. The ability to go fifteen hours without the desire to urinate speaks for an anesthesia of the bladder mucosa common in syphilitic processes of the lower spinal cord. Blunting of the pain sense in the lower legs also speaks for lower cord involvement, as does the disturbance of the bladder reflex. Foot-clonus is indicative

of disease affecting the upper motor neuron, either in the brain or spinal cord. The pyramidal tract is not directly or seriously involved, since there is no suggestion of the Babinski phenomenon and no particular disturbance with locomotion.

The pupillary disturbance—loss of light reflex with preservation of that of convergence—is typical of a syphilitic pupil, but is pathognomonic of it only where degenerative processes in the optic nerve have not gone too far. When they have progressed to the degree found in this patient the sensory arm of the reflex arc is so affected that we cannot expect a reflex.

Multiple sclerosis is to be considered in the diagnosis of any disease affecting definite portions of the central nervous system. Multiple sclerosis, however, is essentially a disease of the second and third decades, rarely coming on as late as thirty-seven, the age of incidence of this man's disease. The cardinal signs of multiple sclerosis, without which we could scarcely make a diagnosis, are entirely lacking; namely, intention tremor, nystagmus, speech and emotional disturbances.

As against brain tumor, as I have already said, the symptoms of increased intracranial pressure are not present. There are no localizing signs suggestive of focal disease of the brain. Disturbances of the bladder sensorium and reflexes are uncommon in brain tumor except where it affects the basilar ganglia or midbrain. If a tumor were present in either of these locations we would surely expect some localizing symptom because of the importance of the contiguous centers—some oculomotor palsies, some disturbance of cranial nerves other than merely the optic nerves.

The positive evidences suggesting syphilis as the cause of the primary optic atrophy are: first, the character of the atrophy—the disk in this case is typical of that found in late syphilis of the degnerative type; second, the bladder disturbance is of the type most often found in syphilis; third, the sharp pains which he had last year are suggestive of those commonly met in locomotor ataxia; fourth, blunting of pain sense is a very common symptom in late nervous syphilis; fifth, foot-clonus without a Babinski sign is also not infrequent in late syphilis.

There is one other disease which must be considered, and that is combined degeneration such as commonly is associated with or dependent upon a pernicious anemia. In that disease, however, the sensory symptoms are more marked than in this patient. Marked paresthesias, weakness, numbness of the legs, are all prominent early in the disease. Increased reflexes are practically always associated with a Babinski sign when they occur. The more frequent picture is the condition opposite to that which we find in this patient; namely, a Babinski sign with diminished deep reflexes. The blood-picture shows no evidence of the pernicious type of anemia, so we can disregard this disease.

Having diagnosed the fundamental trouble as syphilis, the question remains, What is the nature of the process, what the appropriate treatment, and what the outcome to be expected? As for the optic nerve, the syphilis is parenchymatous, is of the nerve-fibers themselves; as such it is beyond our present means of treatment. The prognosis is entirely bad as regards his vision.

The bladder disturbance, associated as it is with some sharp pains in the shins and the blunting of sensation in the lower legs, is probably due to a meningomyelitis of the lower portion of the lumbar enlargement of the cord. This can probably be affected by thorough antisyphilitic treatment. The increased reflexes indicate trouble in the neighborhood of the pyramidal tract somewhere in its course. It is probable that this is of a parenchymatous nature because of the late stage of the disease and the absence of any acute symptoms, such as the sudden giving way of the legs, slight strokes or severe cramps, as in intermittent claudication.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

AORTIC ANEURYSM. THE FACTORS GOVERNING INEQUALITY OF THE PULSES

THE first patient I want to show you comes in with the following history: He is an Irishman, thirty-five years of age, a laborer. It is an old case, one in which the diagnosis is very simple. He comes in for a tumor of the breast. The onset of the trouble occurred two years ago when he noticed a sudden pain in the chest, not aggravated by breathing or by coughing. It seemed to him to be in the ribs; he has had no such pain since. Soon after this a swelling appeared in the sternum; this soon became larger and as it increased in size the heart-beat became more noticeable.

His past history is of some consequence: He has had a urethritis; had a chancre eight years ago, single, hard, followed by secondaries. He says he took blood medicine for several months afterward. He then went to a hospital, where this condition was investigated and a diagnosis of aneurysm was made.

At the present time the diagnosis is so simple that it would almost insult your intelligence to think you could not make it at a glance, but he presents so many interesting features that I decided to bring him down.

Examination of the face discloses nothing abnormal; on the right side of the neck the jugular vein is very prominent, on the left side we can't see it. In the carotids the pulses are full and equal. When we come to the chest we are immediately struck by this tumor. We see the tumor three-fifths to the right of the middle line and two-fifths to the left of it, about as large as a good big fist, extending from an inch below the jugulum to three or

four fingers above the ensiform. It takes in the middle three-fifths of the sternum. In the first place it pulsates—indeed, the whole chest pulsates—and if we get down and look at it from the side we can see that it is a typical expansile pulsation; the excursion is very great—it moves fully $\frac{1}{4}$ inch in every direction. The diagnosis of aneurysm is perfectly plain, and I don't know of anything in the world with which this could be confused at its present stage. The next thing is to see what vessel this comes from. I might mention that this aneurysm was wired once. The first question is to try to locate the heart. When we look in the ordinary position we don't see anything, but when we come around here to the posterior axillary line in the sixth intercostal space we see the apex-beat. These conditions were formerly spoken of as two hearts in a single breast. The apex-beat proper is way down here, and the patient himself takes the aneurysm for his heart. Let's outline it: The apex-beat is in the sixth intercostal space and behind the seventh rib, almost in the posterior axillary line, three fingers wide, circumscribed, and strongly heaving. Now, if we compare that with its normal position we will see that this apex-beat is dislocated fully 5 inches. The reason I want to outline the apex-beat so accurately is that we are deprived of the possibility of outlining the right border; by outlining the apex-beat we can make a fair guess as to where the right border is.

By percussion we can determine the outline of the aneurysm. I don't know just exactly how to describe that dulness to you except to say that the dulness begins below the jugulum and then goes down half-way between the edge of the sternum and the nipple line as you see. The first question to ask yourself is what vessel the aneurysm comes from, and the answer is the aorta. What other vessel could it come from? The innominate possibly, but not probably. It could hardly originate in the arch of the aorta itself with these findings, because the arch of the aorta does not begin until we get further up, and I take it that this is an aneurysm of the ascending aorta which goes right down to the pericardial sac. We have a couple of very instructive *x*-rays of this heart and aneurysm. You can see by these pretty well how

the thing lies. It extends along the right side of the sternum and probably down into the pericardial sac itself. The *x*-ray confirms pretty well our idea of the location. It extends along the arch of the aorta further than we would expect from the clinical findings. You will remember that the arch of the aorta protrudes backward as it goes upward, and in this point the *x*-ray has done

Fig. 80.—Aneurysm of aorta.

something for us that the clinical methods could not do, for it has enabled us to find those parts of the aneurysm which we could not locate on percussion because they lie so deep.

On auscultation we find the following: At the apex a normal first tone followed by a soft diastolic murmur which grows louder as it approaches the edge of the sternum. The diastolic murmur explains why this heart is hypertrophied in the left ventricle, as

we have already seen. I did not mention when we were on the subject of palpation that we find a very pronounced diastolic shock and that there is no thrill present. One thing we will notice in the *x*-ray is that the aneurysm does not show as much as we might expect. This is because they take the *x*-ray from the front and from behind, and it does not look so big as it would from the side. The only way we could show this plainly would be to take it laterally, and that would be very difficult to do.

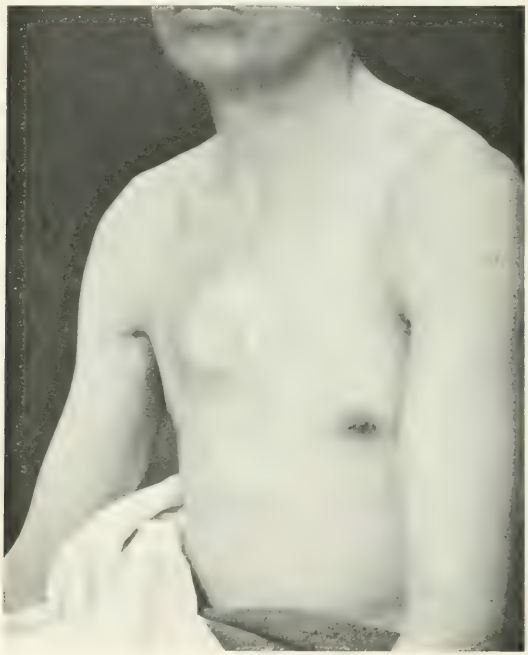

Fig. 81.—Aneurysm of aorta.

Now what do we expect in the way of peripheral signs in an aneurysm located in this position? If we have an aneurysm of a sacculated character what resistance does it offer to the blood? Practically none. If the aneurysm is at one side, the blood goes right by the sac without meeting any impediment; the result is, that with a sacculated aneurysm we may have very little difference in the heart action or in the pulses. We want to emphasize

this because one is constantly expecting changes in the pulse. How can an aneurysm of this kind affect the pulse? The first way

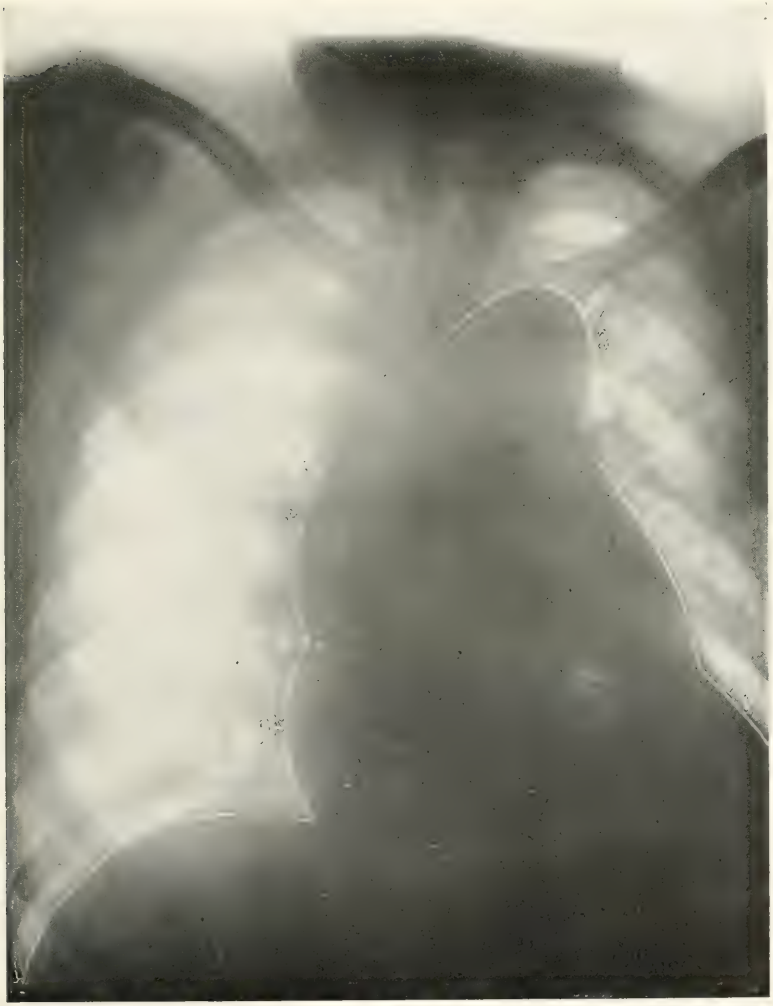

Fig. 82.—Skiagram of aneurysm shown in photograph. Note how much smaller aneurysm appears in skiagram taken anteroposteriorly, hence the bulge does not show here as it does in photograph.

is by extending over such a distance that it draws a large branch, such as the innominate, into the aorta itself, and in that manner

becomes an aneurysm of the innominate as well. The most common way, however, is that as the aneurysm grows it pulls on the blood-vessels and so we find the innominate almost closed by traction. Or, it may do another thing; it may press on the outside. The sac may extend up and make pressure on the innominate from without. But in a surprisingly large number of cases nothing of the kind happens. In a great many cases of this type we find no disturbance whatever in the pulses.

In this case it seems at first sight as if the left radial pulse were a trifle smaller than the right, and the blood-pressure is a few points lower on the left than on the right. I am not disposed to lay any stress at all upon that point. This very slight difference in the pulse is of no consequence, and has, I think, nothing to do with the aneurysm. Is the pulse in the abdominal aorta delayed in comparison with that in the breast aorta? Remember that it takes some time for the blood to flow down to the navel, but if you palpate them simultaneously you will not find much difference between the two. So in my judgment this aneurysm has not produced any difference in the pulses which can be attributed to the aneurysm itself.

The aneurysms that lie in front are the easiest ones to diagnose because they present definite physical signs, whereas the aneurysms that lie deep give few physical signs. What are some of the symptoms and signs which denote an aneurysm of the aorta in any portion? In the first place, pain; pain may be due to pressure on the posterior surface of the sternum; it may be, as it was in this case, referred to the ribs and be due to irritation along the intercostal nerves; it may be due to pressure on the vertebral column, or every now and then we have pain radiating along the brachial plexus. Along with these we have a lot of other subjective symptoms. Patient gets short of breath; this may be due to several different causes—pressure on the trachea, pressure upon the bronchus, or pressure upon the lung itself. A very important symptom is hoarseness due to pressure upon the laryngeal nerves. It would be futile for me to enumerate all the symptoms that may be caused by an aneurysm. Any symptom which is capable of being produced by pressure upon intrathoracic

structures may be due to an aneurysm, depending entirely upon its location. The important thing to bear in mind is this: The aneurysms which start from the first portion of the aorta produce very early symptoms; if they spring from the convexity of the aorta itself they generally occur to the right of the median line; if they spring from the concavity, they may present to the left of the midline. As long as the aneurysms are fairly far forward they show all the symptoms which we are accustomed to find as the result of pressure, and as soon as they get deeper they give practically no symptoms at all.

A patient may go along with a chronic cough, which is diagnosed as bronchitis, for possibly four or six months, until some one wakes up to the fact that the cough is due to an aneurysm on the under surface of the arch of the aorta. Look out, then, for the aneurysms on the under side of the aorta; they are the important ones.

It may be that the aneurysm has involved the ascending portion of the aorta, so that the aortic ring is dilated, but it is more probable that syphilitic infection has produced a syphilitic aortitis, and we have an aortic insufficiency of specific origin. That is a common state of affairs. The aortic insufficiency explains why the heart is dilated and hypertrophied. It is not dilated to the extent that you would think from the position of the apex-beat, because it is pushed out 4 or 5 inches by the aneurysm.

The underlying condition is the syphilis which has produced a mesarteritis. As a result of this we have an aneurysm about as big as a large grapefruit. The outlook is not particularly brilliant. If the operation, which was done by one of our best surgeons, has had no effect, it is not likely that any further surgical interference will have much effect. We will have a protection of some kind arranged for him, for if he were to fall and strike on his chest the aneurysm would almost surely rupture.

The general belief is that in cases of aneurysm death is almost invariably caused by rupture, but according to my experience this is not true. They often develop a chronic interstitial pneumonia, sometimes with pleurisy; some of them die of exhaustion as a result of pain; many cases die of chronic dilatation; the

heart dilates greatly, edema develops in the feet, the liver swells, and they die of a broken-down heart. So I should say that fully one-half of the aneurysms do not die of rupture, they die of exhaustion as the result of pain and sleeplessness, of chronic indurative infections in the lungs; they die of secondary affections. So you must not imagine that every case comes to an end by rupture of the aneurysm. If rupture occurs in this case it will almost surely rupture on the outside, and that is probably what will happen unless we succeed better with his case in the future than we have in the past.

TUBERCULAR PLEURISY. ITS INSIDIOUS DEVELOPMENT. LÖFFLER'S METHOD FOR DEMONSTRATING TUBERCLE BACILLI IN SPUTUM AND EXUDATES

Last week I showed you an instructive case of gout, telling you that the patient did not come into the hospital with the gout. In the meantime we have worked up the case pretty thoroughly and discovered that he presents some interesting symptoms in another place. This is the patient in whom we found many tophi in the ears, but he has not many left, for he has been investigated by several individuals. You will remember that he had pain in the chest below the costal margin, spots before his eyes, vomiting, cough, chills and sweats, diarrhea, and dyspnea. All of these things in addition to the rheumatism, which turned out to be gout. You see he has a large number of symptoms and most of them are referred to the thorax. On going over his chest we find the following: The left side is somewhat more protuberant than the right; Litten's sign is absent on the left; expansion is slight on the left, but present on the right; on percussion the entire left chest shows dulness amounting to flatness over the lower portion, and on auscultation on the left side the voice sounds are absent excepting along the vertebral margin, where we get bron-

chial breathing; on the right side we find nothing abnormal except a few fine cracking sounds at the end of inspiration. These findings indicate pretty well the presence of fluid in the chest. I will demonstrate these points to you. The signs now are not quite so marked as they were a week ago because we have been in there with a needle. He was punctured the first time on the day of our last clinic and we removed 50 c.c. of fluid, of amber color, specific gravity 1017, with a considerable quantity of albumin. On examination of the sediment it showed a good many polymorphonuclears. Smears showed nothing abnormal; cultures showed it was sterile. We inoculated a guinea-pig, and up to date he seems rather to enjoy it than otherwise. The next day we aspirated again and took out 56 ounces for therapeutic purposes. The fluid was slightly turbid, amber colored, very few lymphocytes, and a good deal of albumin and no sugar. The smears and cultures were sterile. Fluid of 1017 points to an exudate rather than a transudate. It came on very gradually, you will remember the whole disease started somewhat gradually. That would point rather to a tubercular pleurisy than anything else, especially as he is running a little temperature. On the other hand, we usually find a great excess of lymphocytes in tubercular pleurisy, but here we do not.

The white cell count showed 15,000 to start with, which has dropped to 13,000, so he is running a slight leukocytosis. The majority of the cases of tubercular pleurisy do not show leukocytosis; very few show over 15,000. This case is about on the fence. If the leukocyte count points either way, it points more toward it not being tubercular than tubercular. Several things point toward it being a tubercular fluid and several against it. The overwhelming majority of cases of primary pleurisy are tubercular. This patient is a great big, strong-looking fellow, the last in the world to have tuberculosis. On the other hand, it was a very gradual onset with no history of pneumonia behind it, and, as I said before, the majority of such cases are tubercular in character. So my recommendation is to adopt the tactics of the "man from Missouri," and that is to take it for granted that it is a tubercular pleurisy until you can demonstrate that it is not.

The sputum was examined and found negative. If we wait long enough the inoculation of the guinea-pig will tell us, but that takes three or four weeks. The common way to inject a guinea-pig is to take a few drops and inject intraperitoneally. In cases that are known to be definitely tubercular if only 1 c.c. is used it will not kill the guinea-pig, so I recommend you to use large amounts, say, 20 c.c., and in that way the fluid in 65 per cent. of the cases of tuberculosis will kill the guinea-pig. The great objection to the guinea-pig inoculation is that the patient is gone from observation before the tuberculosis can be demonstrated in the pig.

There are many ways of finding the tubercle bacillus in such cases: the first method is to make a smear, but there is so little chance of finding the bacillus that it is hardly worth doing. The way I prefer is the **Löffler method,** and in this case we used that method and had no difficulty in finding tubercle bacilli. I will give you the technic of the Löffler method, first in sputum. Take the entire mass of sputum, if it is not too large, and mix it with an equal quantity of a 50 per cent. solution of antiformin, and shake thoroughly, put into a Jena glass flask (which will not be attacked by the antiformin), and bring it to a boil. The sputum goes into solution very promptly and you get a fluid that looks like a turbid urine. *The antiformin must be absolutely fresh.* It has the ability to dissolve everything except lipoid substances. All the bacteria with the exception of the acid-fast group are destroyed by this process. Allow it to cool, and then take 13 c.c. of this solution and put it in a glass-stoppered cylinder, and to this add 2 c.c. of a mixture of 9 parts of alcohol and 1 part of chloroform. After you add to the 13 c.c. of sputum-antiformin solution the 2 c.c. of alcohol-chloroform mixture you shake very violently to make an emulsion; then put into a centrifuge and revolve very rapidly. One should have a high-speed centrifuge that has not less than 3500 or 4000 revolutions per minute. The result of this is that in the bottom of your centrifuge tube you get a little bit of chloroform and on top of that a little bit of a disk, and above that you get a supernatant clear fluid. The best tubes to use are those which open at the bottom. The beauty of

this method is that you can pull out the little cork and let the little pellet drop right out. By using these tubes I found that I could get the bacilli in a much larger proportion of cases than I ever could by smears. In a fluid of this sort (pleuritic) there is not so much albumin as in the sputum, so I recommend that instead of using one-half you use one-third or one-fourth antiformin solution.

This method I regard as one of the most important that I know of. I have already gone on record as saying that I have had more satisfaction out of it than almost any other method in routine laboratory work.

There are several pitfalls. In the first place, the antiformin may not be right. One very distinguished gentleman wrote me that he boiled sputum with antiformin and it got as firm as a hard-boiled egg. His antiformin was worthless. The antiformin must be absolutely fresh; get it in small bottles, keep it away from the light, and you will have no trouble. The second point is to shake very thoroughly with the alcohol-chloroform, so as to make an emulsion, and then centrifuge for at least fifteen minutes at the highest possible speed, at least 3500 revolutions per minute. It is not at all the same thing to centrifuge for two or three hours at a low speed. Pleural fluids are among the hardest fluids we have in which to discover tubercle bacilli.

We will demonstrate the test for you on the sputum. This is the sputum to start with; we simply mix this with an equal volume of 50 per cent. antiformin (which is made up fresh), and put the mixture in a Jena glass flask. You will see that the sputum goes into solution without much difficulty. The principal value of this is to be found in thin watery sputum where the bacteria are scarce. Another great advantage is that when you get through you are examining a sediment in which they are to be found readily. (I don't want to be understood as saying that you can find tubercle bacilli in a large majority of pleural fluids, because you can't. But you can find them comparatively often.) Now you see our sputum is clearing up and has gone into solution; now take 13 c.c. of it, allow it to cool a little bit, add 2 c.c. of alcohol-chloroform mixture, and then shake violently. Now

just imagine that this has been taken up stairs and centrifuged, and this is what you get (holding up a tube of centrifuged sputum).

These tubes are the ones which I use and which were described by me two or three years ago. You can see the little pellet which contains the bacteria. There are one or two little tricks in the staining that I want to show you. You pour off the supernatant fluid from the top of the tube, take out the little pellet and put it on a slide, then add a little egg-albumen, for all the sputum albumin has been dissolved; then fix it by passing through the flame, and stain it in the ordinary manner. I prefer to use the little metal dishes which have little elevations on the bottom to prevent the slide from burning, because I like to boil the specimen thoroughly. In cases where there is the slightest question about the fluid I recommend you to boil it very thoroughly for five minutes and take it out and decolorize it in any way you like. I prefer the acid-alcohol. I think the nicest counterstain is the malachite green solution. All you have to do is to pour off the supernatant fluid; you see the little pellet which looks like a hypodermic tablet, draw out the cork from below, tip the tube a little, and it comes out without further difficulty. You have all the contents of that sputum in this small quantity; do not forget to add a drop of egg-albumen or all you will have to look at will be the blank slide, for the antiformin has destroyed the albumin. Make a pretty thick smear, because it is quite transparent, let it dry in the air for a minute or two, run it through the flame in the ordinary way; now put it into the little dish, cover it with carbol-fuchsin, and let it boil.

Now as to the care of the tubes: wash them out and then place them in a strong solution of sulphuric acid and bichromate of potassium. I keep tubes in this all the time, and when I want to use them I simply take them out, run a little water through them, and run no danger of leaving anything sticking to the tube. You can see under the microscope the bacilli from this case.

We find that this man, aside from his gout, has a tubercular pleurisy. Many of these cases of tubercular pleurisy owe their origin not to the lungs, but to the bronchial glands. We don't

show you this case because we think it is a great feat to find the tubercle bacilli in the pleural fluid; the method is not one which should be regarded as at all difficult; it is a fairly simple one. In the differentiation I recommend you to use acid-alcohol, for then you have not only the acid but the alcohol, and when you get through you have proved that the bacilli are not only acid fast, but alcohol fast. If you boil vigorously for five minutes you need have no fear of overdecolorization. I recommend the method to you in very high terms. I do not know in what proportion of specimens you can find the bacteria, but I know you can find them in a fairly large percentage.

TUBERCULAR PLEURISY FOLLOWING A PERIURETHRAL ABSCESS

This patient was sent over from the surgical department and is a very interesting case. He is single, thirty-six years old, a sewer builder. He came in for a periurethral abscess, was operated on some little time since, and a large amount of pus was removed; the case was going along very nicely until one day the patient suddenly developed the following: A temperature running up to 103° or 104° F., with a chill and pain in the right side of the chest; the findings were such that the case was diagnosed as pneumonia, and he was transferred to the medical service.

When he came into our ward ten days ago he had a temperature of 100° F., and has been running a very irregular temperature ever since, from 100° to 102.5° F. At present it is 103.4° F.

The physical examination on admission to our service (after being in the surgical ward for several weeks) was the following: the expansion over the lower portion of both lungs poor; there was a slight increase in the whispered and spoken voice sounds, and small crepitant râles scattered over the chest. There was a very slight dulness on the right side. Those are not the findings of

pneumonia, and although he was transferred on that diagnosis, they did not really regard it as a pure pneumonia. We did a good deal of laboratory work: Five different blood-cultures taken at intervals of two or three days were all negative; the Widal has twice been negative; the leukocyte count was 5600 and it has never exceeded 7000; we did a lumbar puncture and the spinal fluid was clear and colorless, and the Nonne and Noguchi tests were both negative, as was also the Löffler; cultures from both the urine and spinal fluid were negative. After two or three days the flatness over the lower part of the right lobe increased and we were sure there was fluid present, so we inserted a needle and drew it off. As you see, this fluid is tolerably bloody; it is old broken-down blood-pigment which gives it that color. We have punctured him four times and each time obtained this same dirty looking, hemorrhagic fluid.

We have, then, a man who entered the surgical ward with a periurethral abscess, and was getting well, when all of a sudden he develops a fever and large and small râles all over both lungs, indicating a bronchitis. The temperature drags along, now and then going up a little higher. The man became delirious and consequently we were unable to obtain his sputum for a time, but after a number of days we succeeded in securing some sputum and found tubercle bacilli in it. He undoubtedly has a tubercular pleurisy.

This case is interesting in that it shows you how tuberculosis develops in the pleura right under your nose. It was supposed that it was septic, but he had a persistent leukopenia, and when a patient has a distinct leukopenia with a pleurisy you must always suspect tuberculosis; so we kept after the sputum until we finally found the tubercle bacilli. Now, the question is, Was the periurethral abscess tubercular? It was not so considered in the surgical ward, but was thought to be gonorrheal. Under our very eyes, while he was apparently doing very well, he developed this tubercular pleurisy.

The hemorrhagic pleurisies that we see are tubercular in character in the vast majority of cases. A few of them are due to malignant disease in the pleura, and occasionally we find hemor-

rhagic pleurisies in some of the very rare infections. I have seen 2 cases of it on the other side of the water in anthrax.

To sum up, the man came in on account of a periurethral abscess, was apparently doing well, when he developed what was thought to be pneumonia, and he slowly developed some foci in the lungs, with a leukopenia. He had an exudate in his chest, and though we were unable to find the tubercle bacilli in the pleural fluid, we did find it in the sputum. The case, in my judgment, is a perfectly clear one except as to whether the abscess was tubercular or not.

These 2 cases of tubercular pleurisy ought to make two or three points very plain to you: How it develops in big, strong individuals without an apparent cause; tubercular pleurisy is one of the common conditions that develops in hospital practice; one great difficulty is that in a great many cases the tubercular nature is overlooked.

Now one or two words as to what we find at autopsy in these cases: In many instances you can look very carefully and find no microscopic evidences of tubercles whatever, but when you examine the tissue under the microscope you find a lot of very fine tubercles where you could see none at all macroscopically. Some of these cases clear up, adhesions form, the pleura thickens, and if the patient dies later on you find a very much thickened pleura and never dream that it was tubercular, but under the microscope you find the tubercles. So the diagnosis is not only very difficult during life, but even after death it is not so very simple. Take it to heart, then, that the majority of such cases as you see them in a hospital like this, are tubercular. I except, of course, those that follow a pneumonia; but of those that develop without any apparent cause the overwhelming majority are tubercular.

The evidence must present itself to you in this way: You are to regard a case of primary pleurisy with effusion as tubercular until you can prove that it is not. In some of the cases you will be able to find the bacilli in the aspirated fluid, and in some you will be able to find them in the sputum.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

A CASE OF UNCOMPLICATED DUODENAL ULCER. THE DIFFERENTIAL DIAGNOSIS AND MANAGEMENT

THE patient was admitted on the 22d day of March, 1915, with a history which is so typical that it is almost a pattern case. He is a clergyman, and enters the hospital with the following history: He is forty years of age. The history is negative as to tuberculosis and cancer. He has three brothers living and well, two died of measles in childhood; he has two healthy sisters; his father died at fifty-six or fifty-seven, his mother is living and well. The family history is very good; there is nothing in it that has any bearing on the present trouble. He had measles in childhood. In 1893, when nineteen years of age, he had typhoid fever which lasted for four months because of a relapse. It was evidently a very severe attack, but there were, however, no complications. In 1913 he was operated upon for a ruptured appendix.

Present Illness: A year after that, in October, 1914, his present illness began. He noticed a slight pain in his stomach just a little to the right of the midline. The pain became worse from day to day; it usually came on about three hours after a meal and was associated with belching of gas, which was very annoying and which did not relieve him.

In December, 1914, the pains (which up to that time had been dull and aching in character) began to come on a little earlier, about two hours after each meal, and tenderness appeared just a little below the right costal margin. The pain below the sternum was sometimes relieved very quickly, sometimes almost immediately, by taking food. During the month of January, 1915, the symptoms became more marked. By February it was neces-

sary for him to eat every three hours or oftener to keep the pain away. By March the pain came on about an hour after eating and it was necessary for him to eat still more frequently. He made these observations himself and they are much keener than the average individual makes, for many people are not trained to think, and the histories given are not so good as they would be if there were more intelligent observers. Hence, such a history as this is of great clinical value.

When the stomach was empty the pain was very severe, and was always in the same place; a sensation of burning and distention was also present, and was only partially relieved by the belching of gas, which was facilitated by the patient lying flat on his back in bed. Here I will call attention to a little point which I think is very important diagnostically. He noticed that if he ate a heavy meal the pain was delayed longer than when he took a light meal; when he took a heavy meal he might not have any pain for two hours, but after a light meal there was invariably pain after one hour. On the other hand, the pain was often more severe after a heavy meal and less severe after a light one. The patient occasionally experienced a little nausea, but he has never vomited. The pains appeared constantly an hour or two following meals and disappeared quickly on taking food.

From that history it is possible to make a diagnosis in his case as we proceed. We will give you what other information we succeeded in getting. Urinalysis on the 23d of March was negative: Specific gravity 1020, no albumin, no casts, no sugar, one or two white cells to the field, no red cells.

Diagnosis.—The most important symptom which he had was pain; at first it occurred three hours after meals, and gradually the time grew shorter and shorter after the taking of food, so that in March the pain appeared in a little more than one hour after eating. The second point is that this pain was always uniformly relieved by the taking of food. Those two facts in themselves are of great importance. To these are added more findings: First, the history of gas and of burning. These symptoms are not of much importance, because gas and belching, burning and indigestion are complained

of in ulcers of all sorts, whether duodenal or gastric. As to negative points, you will notice in the history that there is no mention made of any colicky attacks, a matter of considerable importance, because in arriving at a diagnosis it helps us to exclude conditions associated with colic. You will also notice in the history that there is no reference made to an infectious process. There was no history of joint disturbance, no history of infectious headaches, no history of a fever or of a chill, and no increase in the leukocytes. Consequently, then, in massing our data for a diagnosis, this additional negative finding is of extreme value—the absence of signs of infection; and, lastly, another very important point, there was no vomiting. In other words, there have been no reflex antiperistaltic waves sweeping bile into the stomach and causing vomiting, nor has there been any reflex or disturbance in the stomach itself which has led to the ejection of food. As a matter of fact, he has felt worse when his stomach was empty and better when it was full. In this respect he is just like a patient we had here last fall, a young physician, who had been treated previous to his arrival at this hospital by gastric lavage. He told us that washing out the stomach made him feel worse instead of better.

Dr. Mix: Now, what has he?

Visitor: Hyperacidity.

Dr. Mix: Hyperacidity is merely a symptom; the burning he complains of makes us believe that he had it all right enough; but hyperacidity is not a disease.

Visitor: Pyloric stenosis.

Dr. Mix: There is no history of a long-continued gastric disturbance such as might ultimately lead to obstruction at the pylorus. His trouble dates from October, 1914. Besides, he has never vomited and his stomach is not dilated.

Second Visitor: Gastric ulcer.

Dr. Mix: We will consider that when we speak of the differential diagnosis.

Another Visitor: Ulcer of the duodenum.

Dr. Mix: Yes; anything else?

Visitor: Tumor of the pylorus.

Dr. Mix: Possible only, for a tumor of the pylorus ought to produce dilatation and vomiting. There are always four things that have to be considered in these conditions, perhaps five, and these five things are gastric ulcer, duodenal ulcer, chronic pancreatitis, gall-bladder disease, and chronic appendicitis. The reason that chronic appendicitis has to be considered is that it frequently produces a set of reflex disturbances at or about the pylorus and frequently gives rise to attacks of pyloric spasm with great epigastric pain. Oftentimes the pain is so severe that these people empty the stomach either by forced vomiting, by using salt and water, putting a finger down the throat, or by washing the stomach out with a tube. I remember an individual who had such an appendix whose sole thought was his stomach. We recognized the stomach symptoms as reflex and supposed them to be partly due to the gall-bladder and partly to the appendix. He was slated for operation to have the gall-bladder drained and the appendix removed. When he came to operation there was found to be nothing wrong with the gall-bladder; it was perfectly negative, and the whole group of symptoms was due to the appendix. Remember, then, that the patient whose sole thought is centered upon his stomach may have his trouble as a sequel to chronic appendicitis.

Of these five things we can dismiss one immediately, and that is chronic pancreatitis. We are not able at this time to diagnose cases of chronic pancreatitis. We recognize several symptoms which come from it—vomiting (sometimes severe), loss of weight, pain, disturbances of color; but making a diagnosis with any degree of accuracy seems to be quite impossible at present.

We can also eliminate the appendix because it has already been eliminated for us; it was removed in September, 1913. His present illness came on thirteen months after the removal of the appendix. So there remain for our consideration only the diseases of the upper portion of the abdomen. Assuming, however, that the appendix had not been removed, in going into his case and examining it carefully, we might have found times when there was a slight increase in the temperature, associated with a slight leukocytosis. We would also

have found tenderness in the region of the appendix because (although appendicitis sometimes exists without spontaneous pain) there is always tenderness and frequently rigidity over the right iliac fossa. Had these signs been present we should have been obliged to say that they existed because of the appendix, and it might be wise to have it removed in the hope that the trouble with the stomach would be thereby cured.

The next proposition is whether or not the gall-bladder is involved. But I think that instead of considering the diagnosis first in this case we can better arrive at a conclusion if I tell you the story of gastric ulcer and duodenal ulcer. The simplest thing to consider is duodenal ulcer: Ulcer of the duodenum is frequently observed in this region, sometimes coming on in its earlier stages with sufficient pain to make its presence at once evident, but oftentimes coming on vaguely and insidiously and not attracting the patient's attention until it has existed for quite a long period of time.

Indeed, when duodenal ulcers began to be diagnosed frequently, a few years back, most of the cases which came to the surgeon had existed on an average of twelve years. Most of these cases used to be called gastric neurasthenia and gastric dyspepsia. The man who deserves the greatest amount of credit for establishing the symptomatology of duodenal ulcer is Moynihan of Leeds. At first the symptoms were much obscured by secondary adhesions and complications, but of late, since the diagnosis has been worked out more carefully, we have learned the earlier symptomatology of duodenal ulcer, and it is surprisingly exact and very simple.

Duodenal ulcers in some people are said to be more apt to appear in the spring and fall of the year. This man's case bears out the rule, his onset being in October. The exacerbations are more marked in the spring and fall. In the midwinter and midsummer duodenal ulcers of the chronic type get along fairly well.

The etiology of duodenal ulcer has been stated by Rosenow, of this city, to be an infection, and by injecting dogs with streptococci of a certain virulence he has been able to produce duode-

nal ulcer in varying degrees. The old theory is that such ulcers are due to the occlusion of a terminal blood-vessel by an embolus; the tissue, being deprived of nourishing blood, is destroyed and the ulcer forms. The fact that most ulcers are located very near the pyloric artery, on either the gastric or duodenal side, lends belief to this embolic idea.

Support is still further lent to this theory by reason of the fact that individuals with duodenal ulcer frequently develop gastric ulcer, and that individuals with gastric ulcer ofttimes develop duodenal ulcer. So anyone who is careful in the diagnosis of an individual case must consider the possible presence of a duodenal ulcer if there is a gastric ulcer, and vice versâ. Usually, if an ulcer appears on the gastric side of the pylorus, which was preceded by a duodenal ulcer, the symptomatology markedly changes. The change in symptomatology is much less spectacular when a duodenal ulcer is added to a gastric ulcer. It is far more difficult, when the gastric ulcer develops first, to detect the advent of a duodenal ulcer, than it is to diagnose a gastric ulcer following a duodenal ulcer, unless one ulcer heals before the other develops.

The cardinal symptom in a duodenal ulcer is *pain*, and it bears a very definite relationship to the meal. Pain comes on a long time after a meal (the text-books say from one to four hours). This patient at the onset had pain three hours after eating, and then, as the case progressed, the time limit fell to two hours, and later still to one hour. There is possibly some relationship between the location of the ulcer and the time of the pain. The observations which we have made in that respect are these: The nearer the ulcer to the pylorus, the sooner the pain appears; the further along the duodenum the ulcer, the later the pain. Moreover, in this particular case, another very interesting symptom appears; that is, with a full meal the pain occurred two hours after, and with a small meal it came on one hour after.

What is the cause of this pain? No explanation has ever been made which has been universally accepted. A favorite old explanation is that the pain is caused by the bathing of the

ulcer in the free and excessive hydrochloric acid of the stomach. Our patient has a hyperacidity and, therefore, presumably has a good deal of hydrochloric acid bathing the surface of the ulcer. True it is, that if the ulcer is in the stomach and acid is ingested, the pain is very much worse; consequently, if the ulcer is in the duodenum, when the acid in the stomach reaches a certain degree of acidity why should it not be capable of producing pain in the duodenal ulcer when it passes over it? Because the acid in the stomach, no matter what its degree, is just about neutralized by the alkalinity in the duodenum, so that when the acid from the stomach flows into the duodenum it is immediately deprived of its sting. Hence, acid gastric juice does not probably explain the pain of duodenal ulcer unless there is an imperfect neutralization of the acid. So far as we know that is not the case, and we shall have to look for another cause of the pain.

A clinical fact is, the nearer the ulcer to the pylorus, the earlier the onset of the pain. Another curious fact is that even when the ulcer is on the gastric side of the pylorus, within the antrum pylorici, pain does not occur until half an hour after a meal and sometimes not for an hour. So you sometimes have delayed pain even in gastric ulcer. These two facts lead one to another explanation and perhaps one in which you can see some plausibility. According to this theory the pain is due to a pinching of the pyloric nerve supply by spasm. If a man has a *fistula in ano* he does not have the pain when the sphincter is relaxed, but afterward, when tenesmus ensues; in other words, when the sphincter becomes very tightly contracted and squeezes the fistula. The pain develops twenty to thirty minutes after the bowel movement, because at that time the pinching of the terminal nerve filaments takes place. The pain in duodenal ulcer may be explained in much the same way. When the pylorus is wide open there is no pain, but when the pylorus begins to shut down after the meal then pain follows. If the pylorus is kept open by taking a large meal, then no pain appears for two hours; but if a small meal is taken and the pylorus is kept open only a short time, then the pain appears

sooner. You know that if you put a quart of milk with bismuth into the stomach for the purpose of an *x*-ray examination, it goes through the pylorus very rapidly, and as long as the pylorus remains wide open there is no pain, but when the pylorus again contracts the pain comes on. That explains to us another most important symptom in duodenal ulcer, namely, the phenomenon of food relief in duodenal ulcer. Why? Because the taking of food relaxes the pylorus and so puts an end to the pinching. How would it work on the gastric side? The taking of food would not relieve the pain of gastric ulcer, because in gastric ulcer the pain is not due so much to contraction of the pylorus as it is to the irritating effect of the food itself and of the associated hyperacidity.

There are some who say that it is impossible to distinguish an ulcer of the duodenum within $\frac{1}{2}$ or 1 inch of the duodenal side and a gastric ulcer within $\frac{1}{2}$ or 1 inch of the gastric side of the pylorus; that if operated upon the ulcer is often found to be, or is quite as likely to be, on the gastric side as on the duodenal. If the explanation that pain is due to contraction of the pylorus is correct it would explain the difficulty in making a diagnosis in ulcers in this region closely adjacent to the pylorus. Because, you see, if an ulcer is situated within $\frac{1}{2}$ inch on either side, the pain would cease when the pylorus is open. For my own part I believe that the differential diagnosis can be made by careful consideration of the pain with reference to the size of the meal and to the time of the occurrence of the pain.

By way of summary, we now know, first, that duodenal ulcers are most apt to appear in the spring and fall; second, that the pain comes on within two or three hours after a meal, and third, that the pain is relieved by the taking of food. Now we must add two or three more facts. One interesting symptom is the presence of tenderness; you can differentiate between colic and ulcer by the tenderness. If you have a colic it is relieved by pressure, but if you have an ulcer of the duodenum it hurts the patient to press upon it. This is a valuable point to remember—sharp pressure elicits tenderness and always over one point, namely, the *duodenal area;* that is to say, about

1½ to 2 inches above the navel and 1½ inches to the right of the midline. Deep pressure in this area reveals tenderness and a feeling of soreness on the part of the patient. Furthermore, the outline of this area is not sharply demarcated; we can't say that it reaches to this point or that point; on the contrary, it shifts off very vaguely, and the reason is that the location of the ulcer is so deep you can't palpate accurately. The tenderness is only to be found through the anterior abdominal wall. It is true that pain in duodenal ulcer is often complained of in the back; indeed, about 35 per cent. of duodenal ulcer cases complain of pain in the back as well as in the front. This patient never had any back pains, but he has had tenderness over the duodenal area. This point of tenderness has about as much value from the viewpoint of diagnosis in duodenal ulcer as McBurney's point has in the diagnosis of appendicitis.

Another symptom of great value and often pathognomonic, is hemorrhage from the bowel. In the examination of a patient never omit careful questioning as to the possibility of such hemorrhage. Never ask your patient whether he has had a hemorrhage, or even whether he ever passed tarry black and stinking stools. Ask him first whether he ever passed a peculiar looking bowel movement. Do not lead him. If, without leading, he accurately describes such a movement, you can be certain of your diagnosis. Some years ago we had at Mercy Hospital a man showing the history of duodenal ulcer. His gall-bladder had been previously opened and drained by some surgeon without the slightest relief of his symptoms. When I questioned him as to his bowel movement he at once brightened up and told me of such a movement on a certain Sunday forenoon. He had gotten up early to go to mass, and was taken with a feeling of great faintness so that he could not go, and later in the day passed a black, tarry, mushy, sticky, and stinking stool. The next day he passed some more of the same sort. Then he passed no more, but he remained pale for some time. Could a description be more convincing? When I asked him whether he had told this to the surgeon he said, "No." "Why not?" I asked. "Because he never asked me,"

he replied. The patient never knew the significance of this fact and he had never correlated it with his other symptoms. Hence, in telling his story it never occurred to him. Never forget the inquiry as to possible previous hemorrhage in these cases.

When we add to these findings a fifth, and that is constipation, you have practically all of the symptoms of duodenal ulcer. Constipation is almost always present in these cases and, indeed, has some diagnostic value. For example, gall-bladder cases are not constipated, and in making a differential diagnosis between gall-bladder disease and duodenal ulcer the question of constipation ofttimes assumes considerable importance.

Aside from these symptoms in duodenal ulcer there are others which I mentioned—gas, belching, indigestion—which are absolutely worthless from the standpoint of differential diagnosis. The patient will enlarge upon them and you will listen, but they have no value in getting at the meat of the matter, because they occur in pancreatic trouble, in gall-bladder trouble, in appendicitis, and in adhesions; and it is really a waste of time, so far as my observation is concerned at least, to go into these symptoms to any great extent.

In the diagnosis of duodenal ulcer there are *negative* findings of exceeding value; remember that no diagnosis is a good diagnosis which does not consider a case both *pro* and *con*. A duodenal case, pure and simple, ought not to have any jaundice, ought not to have any infectious headaches, no history of alleged rheumatism or gout—these things must all be out of your picture if your case is one of pure duodenal ulcer. Consequently, in any case, if you ask yourself the question, Am I dealing with an infectious or a non-infectious process? you will be able to marshall your symptoms either on the side of the appendix or of the biliary tract if they show signs of infection, or on the side of the pure duodenal or gastric ulcer if they do not show signs of infection. If you will keep this in mind you will be able to diagnose cases very much better than you ever did before.

As to the laboratory and *x*-ray findings in duodenal ulcer: In 75 to 80 per cent. we find a hyperacidity. A few cases show a normal acidity and perhaps 4 or 5 per cent. show a hypo-acidity.

Furthermore, the presence of blood in the feces is not infrequent in duodenal ulcer, occurring in 20 per cent. of all cases at the first examination. For this test to be of value the patient's diet must have meat excluded for a period of three days or more, and it should be the rule that the examination for blood should not be made close to the time when bismuth is given for the *x*-ray picture or fluoroscopic examination. As I said, 20 per cent. are apt to show blood in the feces the first time you examine them; 80 per cent. will not. They probably all bleed, but they are likely not to be bleeding at the particular time of your examination. Could you follow the cases for days, months, or even years, you would probably find the bleeding in every instance. Hyperacidity is so constant that Moynihan says in his book *hyperacidity is duodenal ulcer.* It isn't, but it is a fact that a majority of cases of hyperacidity are apt to be duodenal ulcer.

Now as to the *x*-ray examination: This shows one point of considerable importance, and that is the absence of the duodenal cap. There is in a normal skiagram a narrowing of the bismuth shadow at the pylorus and then a duodenal cap just beyond, but in the skiagram of a duodenal ulcer this cap is not present. We think it safe to say that if the cap is present the chance of there being a duodenal ulcer is very slight; if the cap is absent we can say that the duodenal ulcer may or may not be present. If, however, we suspect duodenal ulcer to be present and find no duodenal cap, then we are somewhat more confirmed in our idea. The fluoroscopic information which is derived in these cases I think I hardly need go into. It may be worth while when all examining physicians are equipped with fluoroscopes, but until we all have them the information is useless to us. We all have to get along with our history, our physical examination, and our laboratory findings; some of us may have skiagrams made, but for most of us fluoroscopic examinations are out of the question.

Gastric Ulcer.—Now I want to review briefly the symptomatology of gastric ulcer. As to the season of year, there appears to be no favorite time for its occurrence; it may appear at any time so far as the observations of most physicians go. Sex is

of no importance; neither are the symptoms of indigestion, of hyperacidity, of belching, and of gas, because these things occur in all of these abdominal conditions, but the pain is important. The pain in gastric ulcer comes on quite soon after the meal; often while the patient is eating. A patient sits down to eat, the pain comes on and he stops eating, or he does so in the great majority of cases. Many victims will not eat at all because they know that as soon as they do there will be pain. The duodenal ulcer patient wants to eat because he feels better when something is in the stomach; the gastric ulcer patient does not want to eat because he feels worse when something is in the stomach. The duodenal ulcer patient does not lose much weight; the gastric ulcer patient loses a great deal of weight. The duodenal ulcer patient does not lose weight because he eats frequently to relieve his pain; the gastric ulcer case loses weight because he foregoes food to relieve his pain. Moreover, as I have said, the gastric ulcer pain appears immediately or soon after a meal, whereas the duodenal ulcer pain appears one to four hours after a meal.

Now as to the tenderness: In the gastric ulcer patients it is nearly always epigastric and almost always exquisitely localized. By perpendicular percussion it is usually possible to localize it; quite unlike the pain in the duodenal area, which is vague and diffuse, this epigastric pain is usually localized and sharply outlined. Often you can cover the painful area by half a dollar or even a quarter. Third, is the presence of vomiting. If an ulcer of the duodenum is associated with vomiting it is because that ulcer is trying to perforate or because it has set up a secondary peritonitis; but in a gastric ulcer the stomach usually tries to expel the food and acid which is causing the pain. Ulcers of the lesser curvature are very apt to produce considerable vomiting. That which comes up is rarely or almost never bile; it is either food or gastric juice. There is no antiperistalsis sweeping bile into the stomach. If you wash out the stomach you don't get bile, but a little mucus and sometimes a little traumatic blood. So vomiting in gastric ulcer takes place only until the food and acid are removed.

Another symptom is hemorrhage. This takes place in both duodenal ulcer cases and gastric ulcer cases, and the blood in both instances goes both ways, but gastric ulcer patients are more apt to vomit blood and duodenal ulcer patients are more apt to show blood in the stools. You can't, however, interpret the presence of blood in the stool as conclusive evidence that the ulcer is in the duodenum, for some of the blood from gastric ulcer may flow through the relaxed pylorus and be passed. On the other hand, in duodenal ulcer, the blood may flow into the stomach from the duodenum and be vomited. But the presence of ulcer in one place or the other is settled beyond the possibility of a doubt by hemorrhage.

When hemorrhage takes place from an ulcer it is usually a large hemorrhage. When blood is present in the feces, they are usually black, sticky, and very foul smelling; the patient will tell you very quickly if you make inquiry about the matter. When a patient vomits blood it is also usually a large amount. The little streaks of blood in mucus occasionally vomited by patients and occasionally removed by the physician on passing a stomach-tube are entirely without significance. When a duodenal or a gastric ulcer bleeds, it bleeds hard, and much blood goes into the bowel or much blood is vomited up. The patient usually has no trouble in recalling the fact.

So much as to hemorrhage, a symptom that is often of extreme value in making the diagnosis. Many a patient has the history but will not give it to you spontaneously, because he does not always know that there is a relationship between it and the trouble in the upper part of the abdomen.

The other symptoms of gastric ulcer are much the same as those of duodenal ulcer, and there is not any great amount of differential information to be gotten from mere laboratory findings alone. The diagnosis must be made from a knowledge of the symptomatology, from examination of the patient, and possibly from the presence of minute findings of occult blood at times. It is likely that the number of gastric ulcer cases appearing at clinics are probably $7\frac{1}{2}$ or 8 per cent. of all gastric cases, so common is it. The negative symptoms which I spoke

of in regard to duodenal ulcer also apply to gastric ulcer. There is no vomiting of bile, no headache, no gout, no fever, no chill —these things are all out of the picture.

In **disease of the gall-bladder** (and I speak of this because we want to consider all three of these diseases of the upper abdomen) the history is quite different. There is pain just as in ulcer, whether gastric or duodenal, but the pain is usually beneath the right costal border. It often radiates around to the back, and it is frequently complained of as running up into the right shoulder. Why? Possibly by reason of the reflex neuralgia of the phrenic nerve, for this nerve contains sensory as well as motor fibers. Furthermore, if when examining these cases you have the patient lie flat on his back, and have him take a deep breath, and as the liver comes down you strike a sharp percussion blow over the gall-bladder area you will produce a good deal of pain. The patient will manifest evidences of this pain very emphatically. Where the gall-bladder is somewhat distended the pain caused by such a blow is quite severe and the sting lasts one or two minutes or more. If the gall-bladder is not distended there will be pain as before, but it will be less intense and it will not last so long.

Biliary tract pain has no relation to the taking of food; it comes on just when it chooses and you can't discover that it is related to anything. It is absolutely without reference to the taking of food; if a patient eats this kind of food or that kind of food, it makes no difference, the pain is there just the same. Moreover, it is a pain coming in attacks which are transitory. In duodenal ulcer the painful period may last for four to six weeks, then the ulcer may heal over and the pain will disappear for a while, but in gall-bladder cases the pain lasts only half a day to two or three days. The people say these are "bilious attacks," and they are right; they are "bilious attacks," but they are due to chronic gall-bladder disease.

These patients vomit, and what do they vomit? Most of them vomit bile. The old idea that gall-stone patients do not vomit bile because the bile is cut off by the presence of a stone in the common duct, so that it can't get into the stomach at all, is non-

sense. As a matter of fact, the bile continues to come down the common duct in most cases, and even in the cases where a stone is lodged in the common duct four out of five times the bile gets by it just the same. Only 20 per cent. of the cases of stone in the common duct show icterus; in the other 80 per cent. the bile gets by the stone. In the majority of all biliary tract infections there is antiperistalsis, the bile goes into the stomach and is vomited. Sometimes the vomiting will relieve the attack the same as it does in gastric ulcer, but often the patient will keep on vomiting until the bile ceases to flow into the stomach. As long as bile gets into the stomach these cases are going to vomit, for the reason that bile is an intense emetic.

Besides these symptoms, which are very different from gastric ulcer, you have the same belching of gas, the same indigestion and distress that you have in gastric and duodenal ulcer, and these symptoms do not get you anywhere. But more important are the signs of infection, especially fever during the attack; there is an increase in the number of leukocytes, although the leukocytosis is usually low in gall-bladder cases, running from 10,000 to 12,000; ofttimes there are chills, sometimes sweats, very frequently headaches, not infrequently pains in various joints, attacks described by the patient as rheumatism. I remember a patient we had in the hospital here last summer with a most beautiful rheumatism secondary to gall-bladder infection. The *signs of infection* are the things that help the diagnosis. We had a patient in the hospital two years ago who had a set of symptoms which looked very much like a disturbance on the part of the stomach because she had belching of gas and persistent vomiting, but she had headaches of severe type. Ulcers of the stomach and duodenum are not infectious and do not produce headaches. So it was decided that the stomach trouble for which she had been treated by a specialist in the east for two months did not exist. She had in her history a slight attack of icterus on one occasion, and on account of this and her headaches and signs of infection we finally concluded that, inasmuch as her history was not typical of either gastric or duodenal ulcer and not typical of appendicitis, her trouble was cholecystitis. This proved to

be the case at the operation; the gall-bladder was opened and drained and the patient was cured of her trouble.

Constipation is not present in gall-bladder disease; the patients are usually regular in their bowel movements; they are either very slightly constipated or not constipated at all. Gall-bladder disease is not a disease of assimilation; patients do not lose weight because of gall-bladder disease, and weight is usually very little interfered with by vomiting.

I have been speaking of gall-bladder disease in the abstract because we have to consider it in that way in the individual case. The first consideration as to the presence or absence of cholelithiasis or stone is the presence or absence of colic. A gall-bladder colic you would think would inevitably mean a stone, but it doesn't, because you can have a most beautiful colic due to the expulsion of mere mucus through the common duct. It causes much pain, but there is no stone present.

There is one diagnostic rule which is of extreme importance, and that is this: If a patient has a colic which is followed by jaundice, the colic was caused by the presence of a moving body or a stone. A colic followed by icterus means a stone; a colic preceded by icterus does not mean a stone. It might mean an inflammation or it might mean a tumor of some sort. The distinction is very easy. If the colic comes first and then the icterus, it is a stone; if the icterus comes first and then the colic, it is presumably an infection or a neoplasm. If colic is present you must decide whether it is due to the expulsion of mucus or a stone —if you can.

Stones are very apt to produce an infection, and if they are located in the neck of the gall-bladder or in the cystic duct they produce ofttimes a very peculiar temperature range which is known as an angular temperature. A patient may have a normal temperature at 11 A. M., at noon he might feel chilly, at 1 o'clock the temperature might reach 102° or 103° F., and at 6 o'clock it might be normal. The next day the process might be repeated, very much as in malaria, for frequently these angular temperature cases closely simulate that disease. The reason is this: if the stone is in the fundus of the gall-bladder you can't

get enough virulent poison poured into the body to present this very sudden febrile reaction. If you get this characteristic reaction you must decide that the infection is in a place very richly supplied with lymphatics, as it is at the neck of the gall-bladder, where a stone often lodges and causes ulceration. The same chills and angular temperature may occur if the infecting agent is in the common duct, or when a cholangitis or pylephlebitis is present.

By way of summary, then, in a case of trouble in the gall-bladder you have an individual who usually complains of a set of symptoms occurring in attacks separated by more or less irregular intervals, these attacks being associated with gas, indigestion, and belching, which have no diagnostic value; also associated with quite a bit of pain, the pain, however, being in no way related to the taking of food and, moreover, a pain which is not necessarily relieved by the vomiting which almost invariably occurs. The vomitus, moreover, ordinarily shows the presence of bile from antiperistalsis. The patient is a well-nourished patient and not constipated, there may be a mild history of icterus; usually in about 52 per cent. of cases there is a history of typhoid fever, in certain cases a history of duodenal ulcer, and in other cases a history of appendicits. These are three great causes of gall-bladder disease in after years. Furthermore, there is a history of infection—bilious headaches, dizziness, vertigo, joint disturbance, rheumatism, gout—all of these things at times are present. The stomach analysis often shows hyperacidity, the same as it does in gastric ulcer and duodenal ulcer, but it never shows anything very characteristic. Blood is not found in the stool and there is no history of hematemesis. The history, you see, is very different from the history of gastric ulcer and duodenal ulcer.

The **treatment of duodenal ulcer** is very simple; it is not medicine. It is a matter of diet and rest. A matter of great importance is that the patient be put to bed, because if he is in bed the tendency for the ulcer to heal is increased several times. Furthermore, the diet must be a restricted one. This patient had at first just milk and arrowroot blanc mange; this for about three weeks and a half. He had the milk day and night, had a

glass beside his bed, so that if pain came on at night he could take some milk. The blanc mange was made with arrowroot. Naturally, he lost weight; during the four weeks of treatment he lost 16 pounds in weight, but he also lost his pain, and he began to heal his ulcer. At first he noticed a little pain on sitting up, but after a while no pain at all. The last week he has been on his feet and "on his feed." That is, we have given him a great deal more to eat. Last week he gained 9¾ pounds, so you see a patient can regain weight very quickly. Now that his ulcer is symptomatically healed over, he is only 6 pounds under weight, and in the remaining weeks of his treatment he will probably get the 6 pounds back.

The treatment more in detail is simply feeding frequently, at intervals of every two to two and one-half hours during the day and night, because these pains must not be allowed to occur either day or night. The pain means a contracted pylorus squeezing the ulcer. We must keep the pylorus patent. If, therefore, you can keep these patients sufficiently free from pain the ulcer will heal. We also give them alkaline water; we put 15 grains of bicarbonate of soda in each glass of water and they may drink all they want of it. They are constipated and it is necessary to give a laxative, and the best thing is the heavy oxid of magnesia. They can take from one-quarter to one spoonful of this two hours after the meal to aid in keeping the stomach alkaline, and frequently that suffices to make the bowels move well. Sometimes it becomes necessary to give it only after one or two meals lest it make the bowels too loose.

Rest is quite as important as the diet. These patients are not healed in six weeks, they are merely started upon the healing process. It takes six months to heal an ulcer, and the same care in the diet must be continued for six months to make the healing perfect. At present this patient is taking cream of wheat, soft-boiled eggs, baked potato, and plenty of milk, but no meat, and we are not going to give him meat for some time, and, of course, no fried foods, because they are not good for such patients.

The other advocated treatment is surgical. Should we at once do a gastrojejunostomy? Many surgeons take the stand that just as the diagnosis of appendicitis means operation, so the

diagnosis of duodenal ulcer means the posterior no-loop operation of gastrojejunostomy. I think you should exhaust medical treatment before you enter upon anything surgical. The gastrojejunostomy is not the first thing to be done—it is the last thing to be done. If the medical treatment has no effect, if the pain is not relieved by any such treatment, if a pyloric stenosis has formed, particularly if life be threatened by hemorrhage, then by all means do a gastrojejunostomy, but if in a perfectly fresh case you should suggest a gastrojejunostomy you would be doing the wrong thing. As a matter of fact, we did not use to recognize duodenal ulcer until it was so old and so chronic that it was impossible to cure it medically. But now that we can recognize it so much earlier, medical treatment is of much more value. The probability is that many cases of duodenal ulcer taken in hand vigorously and early are quite susceptible to cure, and I think just as susceptible as by surgical means. I ask you whether if this patient had been operated on he would be as well to-day as he is now? There is no question but that if he had been operated on he would now be obliged to go away and rest at least two months more before he would be able to work.

The great difficulty is this: So many patients have not sufficient intelligence to do what the doctor wants them to do. We ourselves don't want to have patients giving up their time and staying around a hospital if they don't need to, but when we as physicians think they need this treatment, the patients ought to coöperate with us in our desires. We have to educate the public in this regard just as we had to educate it in regard to appendicitis. After the public learns, as it will, that these early cases of gastric ulcer and duodenal ulcer are very easily healed medically, then gastrojejunostomies will be more rare.

I have said nothing this morning about the association of these three diseases in the same patient, but such associations in the practical workings of things are very common. Often the main trouble is in the gall-bladder, secondary to an old healed duodenal ulcer or appendicitis. You, therefore, must work out the diagnosis from the history. You have, however, no great difficulty in unraveling these cases if you have in mind the

diagnostic elements of simple gastric ulcer, of simple duodenal ulcer, and simple gall-bladder disease. A duodenal ulcer and gall-bladder case may often come together. In the case we have shown this morning you have a pure picture of simple duodenal ulcer, as pure as you will find it in any text-book. It is not complicated by anything. You can easily see that if you keep in mind these simple, pure, uncomplicated pictures of disease, you can take the complex ones and reduce them to their elements, and so make a very careful, positive diagnosis.

(NOTE: June 26, 1915. The patient reported himself as feeling fine, as doing his work, and being without pain. As yet he is careful as to his diet, is taking only very bland articles of food, and is eating six times daily. He no longer eats during the night, for he never wakes up with pain.)

CARCINOMA OF THE STOMACH

History: This patient entered the hospital April 3, 1915. He is sixty years of age and gives the following history: He is married; the family history is negative both as to carcinoma and tuberculosis. *Previous illnesses:* Measles and mumps when a child, malaria in 1875 and pneumonia in 1878. He was then well until 1893, when he first became a patient in this hospital. At that time he had indigestion and stomach trouble—twenty-two and one-half years ago. He remained in the hospital for two months and a diagnosis was made of gastric ulcer. He was discharged restored in health. He went for thirteen years without further illness, from 1893 until 1906, when he was again troubled with his stomach, the symptoms consisting of loss of weight and appetite, belching, and some indigestion; but there was no pain except merely that caused by the indigestion itself, which was more a feeling of discomfort than pain. Treatment improved his condition, and again for two years he was all right until 1908, when he had the same sort of an attack, but more marked in degree.

The condition again responded to treatment. Since 1908 he has been well until the present illness. He denies any venereal disease.

Present Illness: He places the onset of his present illness in October, 1914, when he again began to be troubled with his stomach, with belching, loss of appetite and weight, indigestion, and constipation. Please notice that there was no special pain complained of last October or November. During December he caught a severe cold and had bronchitis, and during that same month he began to notice what we found on examination and then asked him about—an enlargement of some of the glands on the left side of the neck.

In February, 1915, he was taken to the Alexian Brothers Hospital, where he was treated for "incipient pulmonary trouble," as he states. We have not the hospital record, so that we do not know what diagnosis was there made in February, but he says he was treated for "incipient pulmonary trouble and stomach trouble." No other actual information was given either to the patient or to his wife which they can give to us. No statement as to prognosis was given him. He left the hospital in about six weeks, improved in health, and says he "picked up" in the hospital. He took medicine to create an appetite and gained 7 pounds in the hospital, $1\frac{1}{2}$ pounds a week. After being home for a time and making no further improvement, he decided to go to a hospital again, and came here.

March 30th the condition of his stomach became very much aggravated and his general health was worse than at any time preceding. He went to bed early on that evening, but could not sleep; subsequently he had a sort of a chill which lasted fifteen minutes and was followed by fever. March 31st he had a temperature of 102° F. and two or three times during the day a slight chill; he took aspirin without any result. Anorexia and loss of weight became more marked. His nose became obstructed with mucus and he had some nosebleed and also spat up a little blood which he thinks came from his nose. Some external swelling on the left side of his nose became apparent; the patient has always slept with his mouth open, but had no nasal defect until this was

noticed April 2d. The bleeding from the nasal cavity continued, and the patient noticed some shortness of breath. He was brought to the hospital at noon on April 2d.

Laboratory findings: On examining his urine we found a specific gravity of 1017, no albumin, no sugar; many granular casts, twelve or fifteen red blood-cells to the field, using a $\frac{1}{6}$-inch objective; a few bacteria, six or eight white blood-cells to the field. You will note his color is rather yellow; it is the lemon yellow of a severe anemia. A blood examination showed 3,720.000 reds, 50 per cent. hemoglobin, and 3400 white corpuscles per cubic millimeter. You will notice that his blood is cut down in every respect, but that the diminution of his hemoglobin is greater than the reduction of the red corpuscles, thus giving a color index of .74 and indicating a grave type of *secondary* anemia. You will notice also that the white corpuscles are reduced to 3400; this is a reduction that is almost coextensive with that of his hemoglobin. It means a general blood reduction.

This number of leukocytes, 3400, is of great importance from another point of view, and that is when it is correlated with his temperature. When he came into the hospital he was pretty well tired out and had a subnormal temperature of 96.4° F., with a pulse of 80. At 6 o'clock that evening the temperature was 101° F., with a pulse of 100, and at 9 P. M. it was 101.8° F., with a pulse of 108; at midnight, 102.4° F., with a pulse of 100. You will notice a rise of 6 degrees between noon and midnight. It is evident that whatever is causing his temperature is not causing a leukocytosis. Most infections except typhoid with resulting fever are supposed to show a leukocytosis, but this individual has no leukocytosis. Has he an infection or a sapremia? And why has he a grave secondary anemia?

On the 4th of April his temperature varied between 99° F. as a minimum and 100.8° F. as a maximum; his pulse varied between 88 and 100. He had a fairly good day and slept well at night. On April 5th his temperature reached a little higher, the minimum still being 99° F. and the maximum 102.8° F., and his pulse-rate rose slightly, ranging from 88 to 100; on the whole, he felt pretty well and slept better. This morning his pulse was 92, his tem-

perature 99.6° F. His respiratory rate has never been over 20 since he has been here.

Physical examination: The most obvious points in his physical examination are the emaciation and the color. The color, you will notice, is a kind of lemon yellow; it is not an icterus. If you look at the sclerotic coats of his eyes you will see that they are perfectly white. He has no trace of jaundice, but his color is clearly yellow, and it is the type that is associated with anemia and not with icterus. He is not of the type that might be called subicteric. The cheeks are somewhat hollow, and clearly indicate the degree of loss in weight.

Dr. Mix (to Patient): How much weight have you lost since October?

Patient: About 30 or 35 pounds.

Dr. Mix: The loss of weight has been considerable, but it has not been uniformly progressive. In another hospital he gained a few pounds and went away feeling better. On looking at his chest the most noticeable thing is the emaciation; the color of the chest is not so striking as it is on the face. The subcutaneous fat has almost wholly disappeared. There are a few small glands on the right side of the neck, and on the left side there are some very large ones. On the right side of the neck the glands are in the subclavian triangle; they lie rather low for the ordinary tuberculous cervical glands, which usually are found in the superior and inferior carotid triangles. The larger glands on the left side even extend below the clavicle. We can't find any glands below the second left interspace in the chest, but we can find them just above the second rib in the first interspace. On the right side of the chest I can't find any glands beneath the collar bone, though I can find a few in the inferior carotid triangle. Going further down, this armpit on the right side shows no lymphatic enlargement. The same is true of the left side. The glands in the left and right groins are normal in size. Why does he have these low-lying left cervical glands?

Looking at the abdomen, we note first the fulness. It looks rather plump, but this is because it contains a good deal of gas. As the patient lies in the recumbent position the level of the ab-

domen rises slightly above the level of the thorax. When one picks up the skin of the abdomen in order to get at the subcutaneous fat, there is none; it is all absorbed. The feel of the skin over both the thorax and abdomen is normal. There is no apparent stiffness of the skin indicating an underlying peritonitis. If we have an underlying peritonitis, particularly a carcinomatous or tuberculous peritonitis, the skin is apt to lose quite a bit of its elasticity and to become a bit doughy from the slightest possible infiltration of serum, but this skin has preserved its elasticity. The loss of the elasticity is due to the fact that the subcutaneous tissue over the abdomen is slightly infiltrated with lymph, and when these cases are operated upon you can often see the lymph escape when the primary incision is made, and so get a hint of the underlying peritonitis even before the peritoneum itself is cut.

On percussing the abdomen you will notice that on the left side it is rather dull. On the right side, with the exception of this area just above the right iliac fossa, it is also dull. Note how dull it is in the upper abdomen over the surface of the liver. Note that in this bulging area, which is very definite, and which lies between the umbilicus and the ensiform process, it is very dull, although it is bulging. This bulging suggests a mass of imprisoned gas, but when we come to percuss it, it does not sound like gas—it is altogether too dull. The only place where we can find any tympany is in an area just to the left, but close to the midline, extending from the symphysis pubis only as high as the umbilicus, and over to the right from this line a distance of about $4\frac{1}{2}$ inches. Above the umbilicus we find dulness all the way up to the ribs, and across to the left subcostal area, and we find that the left side of the abdomen is dull.

Now it will be interesting, since we have an area of dulness and an area of resonance in the abdomen, to see whether they shift when the patient changes his position. (Patient turns on his right side.) You will note that in the left flank some tympany has appeared, but you will also notice that the tympany noted on the right side is still unchanged. Moreover, you will also notice that the dulness persists above the umbilicus to the lower margin of the ribs.

DR. MIX (to Patient): Now turn upon your left side.

Now we will percuss the abdomen once more. The tympany that appeared in the left flank has receded a little toward the right, and it is dull again in the left side of the abdomen. These findings might indicate to us the presence of free fluid in the abdomen, and, furthermore, it might be inferred that the free fluid does not flow as easily from side to side as it should. If the fluid were perfectly free to flow, then it would flow as in an ordinary ascites; but if adhesions were present the fluid might be encapsulated and so be prevented from flowing freely. So we come to the conclusion that fluid is present, but that it is prevented from flowing freely by adhesions or—something else. I can explain the "something else" by reference to a case. A patient had a mass of dulness extending from 3 inches below the umbilicus upward. It was clearly fluid because it would shift, but it would not go into the right or left iliac fossæ. I called for a stomach-tube and took out fully a gallon of black water from the patient's carcinomatous stomach. So in this case we must ask ourselves this question: Is this fluid free in the abdominal cavity save for some encapsulation, or is it encapsulated in an enlarged stomach, so to speak, rather than encapsulated by adhesions in the free peritoneal cavity? I merely ask the question. You all can answer it when you remember the location and behavior of this area of dulness, and particularly if we can later demonstrate to you splashing of this fluid. Because only that fluid can splash which is in the presence of air or gas.

DR. MIX (to Visitor): Suppose you come down and palpate this abdomen; what do you feel?

DOCTOR: I feel something hard.

DR. MIX: Is it sharply outlined or a little vague?

DOCTOR: It seems to be just an area of hardness.

DR. MIX: That would lead me to believe that you do not find that the "something hard" which you feel has a sharp outline. There are two kinds of tumors which you find in the abdomen: one is a sharply outlined or very well-defined tumor, and the other is a tumor surrounded by a lot of infectious granulomatous

tissue, which consequently has a very vague and ill-defined outline. Many malignant tumors are surrounded by a mass of infectious granulomatous tissue which often begins to develop fairly early. The result is that we really have an area of inflammation surrounding an area of degenerated carcinoma with no sharply outlined tumor mass, but with a vaguely outlined tumor mass; and that is the state of affairs we find in this man. That is to say, there is something in the abdomen at this point (indicating), something which is not sharply demarcated, something which is hard. The question is, Can it be a tumor of the liver? May it happen that the left lobe of the liver comes down in this direction and gives rise to the difficulty? Or is it simply a tonic spasm involving the pylorus of the stomach?

Dr. Mix (to Intern): Now put your hand right there (over pylorus) and tell me what you feel. You can't be in a hurry when palpating these things; take plenty of time and feel of it two or three minutes or more.

Intern: I can feel something, but it is not very definite.

Dr. Mix: No, but you can feel something all right even if it is not very definite; is it definite now?

Intern: It's gone again now; now it has come back.

Dr. Mix: Now see if it goes away again.

Intern: Yes, it does; but I can feel a sort of gurgling, too.

Dr. Mix: Never mind the gurgling for the present. Do you feel a tumor or do you feel a peristaltic wave in the stomach? As I feel of it just now the mass seems to be fading away under my very hand. What do you think about it?

Intern: It's certain that there is something there, but I can't tell just what.

Dr. Mix: May there not be two things present? May there not be both a mass of some sort which you feel, that is hard and thick—woody hard, as it is frequently designated, vague of outline—and also something much more definite; namely, an exaggerated peristalsis of a stomach trying to empty itself, and leading to a spasm of the pylorus? Let us watch for a moment the surface of the abdomen about the umbilicus. What do you see?

Intern: I see a kind of movement.

DR. MIX: In what direction is it moving?

INTERN: It is moving from left to right, and then sometimes it seems almost as though I could see a bit of a movement the other way.

DR. MIX: Yes, I think you are right. It looks just like a wave washing up against a rock of a pylorus, and then being dashed back again. And that is just about the exact truth of the matter. Such exaggerated peristalsis of the stomach, seen so easily here because of the emaciated abdominal wall and the fluid imprisoned in the stomach almost suggesting an encapsulated ascites, is almost pathognomonic of pyloric obstruction. It is the old story—hypertrophy of the left ventricle in aortic stenosis, and hypertrophy and exaggerated peristalsis of the stomach wall in pyloric stenosis. The picture is clear now: A bulging abdomen because of fluid in a distended stomach which is trying to empty itself, but cannot because of a pyloric spasm and because also of an organic pyloric stenosis. We know the pyloric spasm is there because we felt it fade away beneath our touch; and we know the stricture is there because when the pyloric spasm relaxed the stomach did not free itself of its imprisoned fluid, which you also felt gurgling because of the air it contained.

Now we next must come to some conclusion as to the edge of this patient's liver. See whether you can palpate it. Is it enlarged? Where is the lower margin? There is only one way to feel the lower edge of a patient's liver when the abdomen is distended by ascites, an ovarian cyst, a pancreatic cyst, or a dilated stomach full of fluid, and that is by the pushing or thrusting method of palpation with the finger-tips—*stossweise* as the Germans call it. It is not easy to find the lower margin of the liver in this man's case because of the distention.

INTERN: I believe the liver comes down to about a finger's breadth above the navel.

DR. MIX: Try by percussion and see whether you are right. You are getting a tympanitic note about $\frac{1}{2}$ inch above the level at which you thought you felt the liver margin. Your percussion results are right and the lower margin of the liver extends downward to a point about two fingers' breadth above the navel.

The next question that I would like to have you try to settle is whether you can feel any inequalities in the anterior surface of the liver, because if you can do so you might be justified in supposing that whatever the primary difficulty was, there are secondary difficulties in the liver also. Now take your finger and apply the ball of it, flatly, right here (indicating), and tell me what you feel. Do you feel anything hard or anything breaking the apparent edge and surface?

INTERN: Yes, right here under my index-finger.

DR. MIX: Yes, that's just where it is. What is it? Is it in the liver or is it a part of the stomach? How big is it?

INTERN: It feels hard, it is about an inch long, and it has a round border. I think it is in the liver.

DR. MIX (to Patient): Does it feel sore?

PATIENT: Not much.

DR. MIX: Yes, it is in the surface and in the edge of the liver. There is no splashing or gurgling here, and no peristalsis to be seen in relation with it, and it is there all the time. It does not come and go as does a pyloric spasm. It is the most obvious diagnosis that it is a metastasis from some malignant growth elsewhere. We are very fortunate to find such a metastasis out in the open. Our chances of doing so in these cases are very slight—offhand I should say hardly one in ten. The next question that we ought to be very much interested in is what?

INTERN: The stomach, I should say.

DR. MIX: Yes, the stomach; and in what way?

INTERN: The exact border and boundaries.

DR. MIX: Yes, and these can be determined only by percussion and by the *x*-ray. Formerly we pumped air into these stomachs and percussed the outline, but now we give them bismuth and buttermilk and get a permanent record of the gastric outline, especially of filling defects, where we suspect carcinoma. But as this man is a ward patient, the hospital authorities do not furnish him an examination for nothing, and we are forced, just as you will be in many of your patients who cannot afford an *x*-ray examination, to come to a conclusion without this aid. Indeed, in a clinic of this sort we must learn to use to the utmost

the methods that cost the patient nothing; when we arrive at some degree of skill in these cases it will be the easier to arrive at a diagnosis in our wealthier patients who can afford the expense of an *x*-ray examination.

Within our reach, however, are the laboratory findings. We must have some information as to the presence or absence of blood in the feces, because 93 per cent. of gastric carcinomas show occult blood in the feces. Only 7 per cent. do not. As to the examination of the feces, there were found some hard, dry masses and some softer portions, yellowish-brown in color, and some mucous masses. Occult blood was *not* found. Microscopic examination was negative except that many bacteria were found—but this means nothing.

The analysis of the stomach contents is quite different. The patient was given an Ewald meal, consisting of two slices of bread and a glass of water. The contents were removed forty-five minutes later. The fluid portion was rather brownish when recovered; the food particles were simply brown and white and the kind that one would find in an ordinary case of stomach contents where crust of bread and white bread have been given. Chemically, free hydrochloric acid was present, 23 points; the combined acidity was 38; the total acidity was 61; there was no lactic acid, no blood, and no Oppler-Boas bacilli. This does not look like a carcinoma of the stomach because we have here none of the usual stomach findings of a carcinoma; that is, he ought to have in carcinoma of the stomach no hydrochloric acid and greatly reduced combined acidity, and we should find lactic acid present in considerable amounts and occult blood present in the majority of cases, and the Oppler-Boas bacilli as indicating the origin of the lactic acid. None of these things are present. Now, what is the matter with the patient? Has anybody a suggestion as to the diagnosis?

DOCTOR: How is the pancreas?

DR. MIX: I would like to know, but we have no certain means of finding out. No tumor of the pancreas can be felt. There is no sugar in the urine. No signs of a pancreatitis exist.

DOCTOR: It might be a carcinoma of the pancreas.

Dr. Mix: Yes, it might very well be a carcinoma of the pancreas, but his color is not that of a sufferer from carcinoma of the pancreas. Now let's go back to the history. What was the salient point in his history of previous illnesses?

Doctor: Trouble with his stomach.

Dr. Mix: Yes, trouble with his stomach—always trouble with his stomach. The stomach is the thing that bothered this man from the very beginning, starting with a definite gastric ulcer in 1893, from which he recovered. He remained all right for thirteen years, then he gets another attack and that bothers him quite a while, and two years after that another attack comes along, and then last October the fourth attack appeared—1893, 1906, 1908, and 1914—four different times. Now we believe very thoroughly in one general diagnostic rule, and that is that *but one pathologic process ordinarily affects an individual.* It may have many complications, but it is the thing that pursues him through many years, one definite pathologic entity from start to finish. Now the definite thing in this man's case is his stomach.

Another thing that we know full well is that in cases of long-continued or continued and intermittent disturbances of the stomach where the diagnosis of ulcer is well established a carcinoma is apt to be a late development. The old theory of displaced embryonic tissue giving rise to carcinoma I don't believe in. I believe that carcinomas are usually indirectly traumatic. They occur on the lips, on the tongue, in the larynx, at the lower end of the esophagus, in the stomach at the cardiac orifice or at the pylorus. When you get by the pylorus you rarely encounter carcinomas until you get down into the sigmoid flexure and rectum. Three per cent. of all carcinomas are carcinomas of the rectum, and of this 3 per cent. 75 per cent. are easily palpated because they are close to the anus. The number of carcinomas of the intestine as compared with those of the rectum are as 1 to 8; they are usually located where the maximum friction comes—where the trauma comes. All of the localities mentioned are locations of maximum possible trauma or constant irritation. This patient has had a lot of stomach trouble caused by ulcer, and the ulcer, the location of which we can't tell from

the meager data of his history, was probably situated somewhere near the pylorus. As time has gone on the ulcer has been active three times and inactive during several years, but last October it started up again. The result of this recurrent disease is that around the edges of this ulcer carcinomatous tissue has begun to develop.

There is an interesting article by W. G. MacCarthy in the current number of the American Journal of Medical Sciences, which I read two or three evenings ago, dealing with this very relation between ulcer of the stomach and carcinoma. Those who have had the greatest experience believe that carcinoma of the stomach develops on a basis of ulcer of the stomach, really from a substructure of hyperplasia of normal cells. Seventy-five per cent. of these cases originate from ulcer, according to the Mayo brothers. On the other hand, there are some who believe that the number is very much less than that. Friedenwald, of Johns Hopkins, comes to the conclusion that less than 20 per cent. of gastric ulcers eventuate in carcinomas. Probably between these two figures the truth lies, but my own observation is that the percentage is much nearer 75 than 20. In other words, I am of the opinion that the majority rather than the minority of cases of carcinoma of the stomach originate from ulcers.

There is one interesting fact connected with this patient's case, and that is the absence of pain. He has had very little pain. We have been "up in the air," so to speak, in our interpretation of the symptom of pain in gastric cancer, but we think that we have learned that the position of the carcinoma is the main determining factor in the production of pain. At the narrowing of the pylorus a carcinoma is apt to produce a greater amount of pain than one located in the greater or lesser curvature or fundus. The pain at the pylorus is probably due to squeezing of the ulcerated area in the contraction of the pyloric sphincter, just as pain from tenesmus takes place in fissure *in ano*. It is true that latent ulcer of the pylorus exists without much pain, but this is unusual. In general, the nearer the ulcer to the pyloric ring, the greater the pain, though there is always some pain from irritation

of the ulcerated and denuded surface of the ulcer, no matter where it is located.

Looking at this patient's case aside from laboratory findings you would be compelled to say that he has carcinoma of the stomach, with marked anemia and with metastasis in the liver. The duration of the pain has been only about six months; the loss of weight, however, has been considerable—35 pounds. The loss of appetite and the loss of blood has also been very marked, the hemoglobin being 50 per cent. We have early to learn in matters of diagnosis to rely upon our judgment in such cases as this one, and we have a large number of facts to balance up *pro* and *con* before fixing upon a diagnosis. You must always pick out the salient things and stick to them. The salient things in this case are the rapid loss of weight, the rapid loss of blood, the loss of appetite, the enlargement of the abdomen due to a dilated stomach, the pyloric spasm, and the mass that we felt. Against these things we have the laboratory findings of a normal acidity, the absence of blood in the gastric contents, of lactic acid, of Oppler-Boas bacilli, and of blood in the stool. But we must remember that there have been many instances of carcinoma of the stomach with negative laboratory findings. I saw a patient once who lived only three weeks after I saw her, yet the laboratory findings were perfectly negative: I made them myself, so that I know. But we could not make any other diagnosis than carcinoma of the stomach even with negative laboratory findings, because we could feel the tumor. As a matter of fact, quite a large number of gastric carcinomas have been reported showing free acid, no lactic acid, and no blood in either gastric contents or the stools.

The diagnosis of carcinoma in his case is tremendously strengthened by the presence of these enlarged cervical glands in the left side of the neck and especially by their presence beneath the left clavicle. Such a location greatly emphasizes the probability of gastric carcinoma. We ought to remove one of these glands, if the patient will permit us to do so, and examine it histologically for carcinomatous tissue. In giving me his history he states that these glands came six months ago, though he has

had enlarged glands in the right side of his neck even as far back as two years ago.

Going back to the main factor, there is the antecedent history of gastric ulcer, recurrent up to October, 1914, there is marked loss of weight in six months, 35 pounds in all, there is a severe anemia, there is no increase in the leukocyte count, there is an enlarged liver apparently containing a metastasis, there is a pyloric spasm, and excessive gastric peristalsis. All of these things indicate beyond the peradventure of a doubt carcinoma of the stomach. On the other hand, the laboratory findings are against carcinoma of the stomach because there is not present a sufficient amount of lactic acid and blood; there is, moreover, normal acidity and normal combined acidity. Notwithstanding the negative laboratory findings, we are of the opinion that this patient is suffering from a carcinoma of the stomach. His carcinoma of the stomach may not be correlated with these glands in the neck; these right-sided glands, according to him, originated two years ago following a dry pleurisy, which probably indicates that they are tuberculous. But very important to my mind is the presence of the enlarged glands underneath the left clavicle. These are very apt to be metastatic.

Treatment.—What is the best thing to do with this man? That is always a very grave question in carcinoma of the stomach. He is beginning to have some obstruction of the pylorus. Some cases of carcinoma get along very well, almost without a symptom, until the pylorus suddenly becomes stenosed and blocked, and from that time on very little passes through the pylorus. We had a patient in October who always had regarded herself as very well. About the middle of that month, acutely and suddenly, without prodromata, the pylorus became blocked. At once vomiting began and the stomach dilated, so that when we passed a stomach-tube we found a large quantity of fluid in the stomach, about 3 quarts of brownish fluid containing occult blood. This patient declared that he had been perfectly well up to three weeks before he entered the hospital.

Our patient is passing things through his pylorus, but he is passing them with considerable effort, because we can feel be-

neath the finger a little gurgling as the fluid is forced from the stomach into the duodenum, and we can even see the exertions which the stomach makes to empty itself. One of two things can be done: there can be a gastrojejunostomy for the relief of the pyloric blockade, or there can be no surgery at all, for in the presence of metastases excision is absurd. If hepatic or lymphatic metastases are present, operation is contraindicated, and the best thing is to let the fatal issue eventuate as soon as it will. The best thing, then, is to remove some of these lymphatic glands and find out whether they are metastatic before operation should be attempted. We also feel that there are metastases in the liver, but we may be completely wrong with regard to the interpretation of this point, and the patient should have the benefit of every doubt, provided the lymphatic glands are not carcinomatous; that is, an exploratory operation should be performed. If as a result of the exploratory operation our fears of hepatic metastases are found to be well founded, the abdomen should be closed immediately.

Then comes the question of pylorectomy. About 25 per cent. of cases of carcinoma of the stomach produce no metastases; that is, this patient has one chance in four of having no metastases, and there is some chance as to life and radical cure provided there are no metastases. Evidently when this man was in the Alexian Brothers Hospital it was not felt by those who saw him that it was justifiable to say he had a carcinoma. No operation was advised; he gained 7 pounds in weight and felt better. But such gains in weight in carcinoma occasionally occur, even in a carcinoma of the rectum. I had such a case where a man gained 5 or 6 pounds before the carcinoma was excised. He is now in California dying from a recurrence.

There is no *medical* treatment in a case of this type. The only thing we can do is to take care of him by dietary means. You can do no good with the *x*-ray or with radium. There is nothing that we have at our disposal outside of an early diagnosis and early surgery that really offers any hope. Where we have nothing to offer and where surgery might help, one feels justified in advising an exploratory operation.

I regard the temperature in this case as due to the carcinoma. The fact that there is no leukocytosis confirms me in this belief. There being a low leukocyte count, I look upon the temperature as being caused by the toxins of the carcinoma. As a matter of fact, fever is almost always present in late carcinomas. The blood and casts in the urine indicate, of course, a secondary nephritis; they probably do not indicate a renal metastasis.

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